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ORIGINAL ARTICLES

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Editorial

THE GROWTH OF NEUROSURGERY

NEUROLOGICAL surgery has come of age as a specialty in most of the large communities of the United States. This maturity has not been attained without certain vicissitudes during the adolescent stage of development. Throughout this period of growth the preeminence of Harvey Cushing as a scholar, teacher and surgeon was the guiding influence in the furtherance of high ideals in those engaged in this particular field of surgical endeavor. In addition, the teachings of a host of eminent neurologists, the ever-widening sphere of experimental investigation and the contributions of many ingenious surgeons, notably Walter Dandy, have been determinants in the establishment of the enviable standards set for this specialty. The amalgamation of these, as well as other distinctive features of neurology and surgery with particular emphasis on practical approach to diagnosis and scrupulous adherence to Halstedian surgical technic, bids fair to promote teaching that will produce more efficient neurosurgeons for the future.

The gradual formation of national, regional and local neurosurgical societies attests to the serious intent of the increasing numbers of young men entering this field. The senior organization, the Society of Neurological Surgeons, was founded in 1920 and now has sixty-three members. The Harvey Cushing Society was founded in 1931 and now has 140 members. The American Academy of Neurological Surgeons was founded in 1908 and now has forty-three members. Now a local organization, the New York Neurosurgical Society, was founded in 1946 and has twenty-seven members. In addition to these organizations the American Board of Neurological Surgery was established in 1940 and to date has certified approximately 250 candidates. Medical schools have recognized the importance of the teaching of neurosurgery and many of them have given departmental status to this specialty. It has now come to pass that in most schools, instruction concerning diagnosis and treatment of diseases of the nervous system amenable to surgical therapy is being given by the neurosurgeon. Medical graduates of the past decade should therefore be reasonably familiar with our accomplishments as well as our limitations. There are physicians, however, throughout the country who, for one reason or another, have not kept abreast of the advancements being made in this particular field; and it was with the thought that this special number would aid in the dissemination of such knowledge that these eminent authors were asked to contribute to this number.

Only pathologic or pathophysiologic states of the nervous system that lend themselves to surgical therapy have been given consideration. Other related subjects might have been included; however, it seemed best to limit controversial issues as well as the results of many interesting experimental problems.

A major portion of neurosurgical practice, namely, lesions resulting from trauma, has not been properly allocated to those most fitted to assume this responsibility, the trained neurosurgeon. The care of patients with lesions of the brain, spinal cord and peripheral nerves caused by contact trauma should be carried out by those especially trained in this field. During the recent war, a successful attempt was made to segregate the patients with such lesions and the surgical assessment and treatment of these was assigned for the most part to medical officers with neuro-surgical training. These gathered about them younger officers who acquired experience and training especially concerning injuries of the nervous system. As these young medical officers adjust themselves to civil practice, the medical profession at large should recognize the value of their war-time experiences and give them an opportunity to apply this recently acquired knowledge.

While this symposium does not cover the entire field of neurosurgery, it is expected that the reader will find in these articles information of practical value and that this issue will serve as an aid in correctly advising patients regarding surgical treatment of diseases herein discussed.

I wish to extend a word of thanks to each author for his prompt acceptance of the invitation to participate in this symposium.

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Original Articles

REHABILITATION OF VETERANS PARALYZED AS THE RESULT OF INJURY TO THE SPINAL CORD AND CAUDA EQUINA*

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IT is well known that during and immediately after the first world war patients who had been paralyzed as the result of a wound or injury to the spinal cord and cauda equina had a minimal chance of survival and no chance whatever of rehabilitation to a point where they could again lead a normal social and work life. In the following years preceding the second world war, work with civilian prototypes of these patients, together with the development of certain technics for dealing with their peculiar problems, has made it possible to change this deplorable situation. The twenty-four-hour mortality in the author's clinic has been reduced to 8 per cent and the mortality at the end of the first week to 19 per cent. Deaths at the time of discharge from the hospital have been reduced from about 75 to 37 per cent and it is now possible virtually to eliminate all later fatalities. In addition, such patients as will cooperate and have the use of their hands and arms can be promised a normal social and work life limited only by the restrictions made necessary by their enforced use of braces and crutches. This knowledge was first available only just in time for its application to the so-called "paraplegic" soldier.[1] Because of certain characteristics peculiar to the circumstances of their wounding and immediate care, it was possible to study in more detail certain complications of these injuries and to extend the benefits gained by this knowledge to both civilian and service patients. Nutritional problems, for example, that, because of lack of material in the civilian group, could be no more than recognized as in need of treatment, were much more common and required urgent solution in service patients. New ideas and methods of therapy were quickly developed to a high point of efficiency. Bed sores, prevented in civilian patients before methods either could or must needs be devised for their surgical therapy, appeared in service patients in practically every case. They were of such enormous size that surgical closure was not only imperative as a life-saving measure but essential for any degree of rehabilitation as well. Such complications as renal and bladder stones, hypertrophy of the internal urethral sphincter and the effects of prolonged suprapubic and perineal bladder fistulas, could all be studied. Finally, the usefulness of certain surgical procedures[2,3,4] in the treatment of spasm was confirmed and the operations standardized.

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* The opinions expressed herewith are those of the author and do not necessarily agree with those of the Veterans' Administration or the United States Army or Navy.

With the cessation of hostilities, the service patients were transferred to the Veterans' Administration for completion of their therapy. This group not only had to face the same medical and surgical problems that the Armed services had had to (with the exception, of course, of the immediate care of the wounded) but also was forced to assume the burden of rehabilitation as defined above. The alternative was to resign itself to the expense and trouble of providing domiciliary care for these wounded veterans for the rest of their lives. Because such rehabilitation on such a scale was virtually a new problem, certain centers were designated for the concentration and hence more efficient treatment of the "paraplegics." This paper is a brief study of the results of the first year of treatment of patients paralyzed as a result of wounds and injuries to the spinal cord and cauda equina and cared for at Cushing Veterans' Administration General Hospital, one of those centers. It covers the period from October 1, 1946, to October 1, 1947.

For comparison, certain figures in relation to this problem that were collected from five Army and Veterans' Administration paraplegic centers as of August 1, 1946, are interesting. Six hundred ninety-two cases of injury to the cord and cauda equina were analyzed. Sixty-two per cent were paraplegics in the medical sense of the term, that is, had complete motor and sensory paralysis below the level of the cord injury; 3 per cent were quadriplegic, 22 per cent had partial cord lesions and 14 per cent partial cauda lesions. Less than 4 per cent of these patients were reported as ready for discharge from the hospital at the time of the survey. Another 4 per cent were too sick for definitive treatment. Twenty-seven per cent were still in need of neurosurgical operations, 62 per cent were not yet out of bed and 89 per cent were not yet out of wheelchairs. (Table 1.) Even though the survey did not include all the "paraplegics," I believe that the sampling was done in

such a way as to approximate the general picture. Furthermore, in general, because of the time of the survey and the sampling, it can be assumed that this is a reasonably accurate picture of the cord injury problem as it was turned over to the Veterans' Administration from the Army. Table II shows the comparative figures at Cushing General Hospital, one of the centers in-

TABLE I
SURVEYED PATIENTS AUGUST, 1946

	All Cases		Percentage of Patients				
	No.	Percentage Distribution	Too Sick for Definitive Therapy	Still in Need of Neurosurgical Operations	Not Yet Out of Bed	Not Yet Out of a Wheel-chair	Ready for Discharge
All injuries.......	678	100	4	27	62	89	3+
Paraplegics......	418	62	3	30	70	95	0+
Quadriplegics....	18	3	18	0	0	0	0
Partial cord injuries.........	121	22	6	26	52	83	7
Sacral segment injuries.........	22	0	5	11	34	79	0
Partial cauda equina injuries	93	14	1	13	44	70	14

ministration from the Army. Table II shows the comparative figures at Cushing General Hospital, one of the centers in-

TABLE II
CUSHING GENERAL HOSPITAL AUGUST, 1946

	All Cases		Percentage of Patients				
	No.	Percentage Distribution	Too Sick for Definitive Therapy	Still in Need of Neurosurgical Operations	Not Yet Out of Bed	Not Yet Out of a Wheel-chair	Ready for Discharge
All injuries.......	678	100	4	27	62	89	3+
Paraplegics......	84	63	0	6	56	90	0+
Quadriplegics....	0	0	0	0	0	0	0
Partial cord injuries.........	25	21	0	21	42	59	33
Sacral segment injuries.........	5	0	0	0	0	0	0
Partial cauda equina injuries....	46	16	0	5	23	32	36
All injuries Cushing............	135	100	0	10	46	76	13

cluded in the survey, two months before the Veterans' Administration took charge. As will be noted, the figures are better in many respects than those of the entire series. This is directly traceable to the cooperative attitudes of the then Commanding Officer, Colonel Floyd V. Kilgore and his chief of the surgical service, Colonel Horatio Rogers, in having facilitated the establishment of a therapeutic program that had already been well tested in the neurosurgical department of the Boston City Hospital. It was carried out by a group of what amounted to volunteer medical officers who had had specialized training in neurologic, plastic and genitourinary surgery. Their work was coordinated and directed by a single head. This program was started in April, 1946, and by August of that year a situation that had been virtually hopeless at the start, had been changed into one that reflected tremeneous credit on the hospital and its staff. This same group of army officers augmented by two others as well as two naval officers volunteered to continue their services after the Veterans' Administration took over and are only now—after one year and four months—beginning to be replaced.

Between August 1, and October 1, 1946, the "paraplegic" census at Cushing General Hospital rose from 135 to 148. The latter was the number of patients actually on the Paraplegic Service when the Veterans' Administration took over. During the following year from October 1, 1946, to October 1, 1947, there were sixty-four new admissions to this service. Thus the total number of patients treated for injury to the spinal cord and cauda equina during the first year of the Veterans' Administration operation was 212.

There were also forty-three readmissions. Eighteen of these patients returned voluntarily for checking at various periods following their discharge. Two came back after a C.D.D. discharge from the army and three others were pursuaded to return for further necessary treatment after leaving at their own request. Five others

originally left against medical advice. These have since returned for further treatment. Only seven of the readmitted patients had been carried as A.W.O.L. in that they were given leave to go home for a period and failed to come back when their leave was up. Two of the seven have since returned and have been finally discharged for disciplinary reasons; one returned and subsequently left against medical advice; one was A.W.O.L. and returned and left a second time A.W.O.L.; one returned and was then given a medical discharge; two have returned and are still under treatment.

Exclusive of readmissions, there have been ninety-seven discharges during the year and three others have completed their training and are still in the hospital only because of non-medical reasons. Fourteen have completed their ambulation training but need further medical care. Therefore, nearly one-half (47 per cent) have been or are about to be discharged living and able to take their place in the community and 54 per cent have been taught to walk in the first year of the Veterans' Administration operation.

The 112 patients that are still receiving treatment are the group that have presented all along the more difficult therapeutic problems. They include the quadriplegics (fifteen), those with intractable bed sores (forty-two), major genitourinary tract disturbance (fifteen), complicating osteomyelitis (two), a demonstrated need for neurosurgical (twelve) or orthopedic (six) operations that were not apparent until ambulation training had reached an advanced point, spasms (sixteen), amputations (three), disabling fractures (six), incidental neurologic disease (three), fear (one), laziness (six) and an uncooperative attitude (seven). Only sixteen of this group have progressed beyond a wheel-chair existence. Seventy-four are being given primary ambulation training (fifty-eight) or are still bed-ridden (sixteen). Eight are out on leave.

Two patients died before discharge. One had a transection of his lumbosacral cord and died of uremia almost two years after

injury and seven months after admission to the Cushing Veterans' Administration General Hospital. He had renal infection and stones as well as bilateral nephrostomies. The second had his cauda equina partially destroyed. He had bilateral renal stones and infection, a scrotal urinary fistula and a colostomy. He died following a kidney operation two years and five months after injury and seven months after admission to Cushing Veterans' Administration General Hospital. This mortality rate of 0.9 per cent while satisfactory from the point of view of the care given at the hospital is actually of little value from the larger point of view of the mortality as a whole in this type of case.

ANALYSIS OF THE DISCHARGED PATIENT'S CASE HISTORIES

An analysis of the case histories of the 100 patients that have completed their medical treatment and have either been or

TABLE III
CLASSIFICATION ACCORDING TO LEVEL

	Total Cases		Cervical			Thoracic			Lumbo-sacral			Caudal			
	Total Cases	Percentage	Transection	Partial	Radicular	Transection	Partial	Radicular	Transection	Partial	Radicular	Transection or Total Destruction	Partial	Radicular	No Note
Service cases..	100	100	2	6	0	26	4	0	19	15	0	4	21	0	3
Civilian cases..	313	100	29	119	9	42	37	0	6	25	0	0	44	2	0

are ready to be discharged, brings to light certain interesting data that should not be overlooked. To emphasize its significance, I have compared it with similar data obtained from an analysis of a personal series of 313 civilian cord and cauda equina injuries. (Table III.) The two series offer no fundamental differences although they do vary in certain details. In the service group the distribution of cases is preponderantly on the side of transection (Table IV), while the largest civilian group is found in the partial cord injury category. Virtually no

patients were seen at Cushing Veterans' Administration Hospital in the months immediately following their injury. (Table v.) This is a complete reversal of the civilian situation and, as will be pointed out, may have an important bearing on the

TABLE IV
CLASSIFICATION ACCORDING TO PATHOLOGY

	All Patients		Transection of Cord		Partial Cord Injury		Cauda Equina Injury		Radicular Injuries	
	Total Cases	Percentage	No.	Percentage	No.	Percentage	No.	Percentage	No.	Percentage
Service cases..	100	100	47	47	25	25	28	28	0	0
Civilian cases..	313	100	77	25	181	60	44	14	9	4

occurrence of certain signs and symptoms that may be wrongly thought to be characteristic of cord and cauda injuries as a whole.

TABLE V
LENGTH OF TIME BETWEEN INJURY AND ADMISSION

		Less than 24 hr.	1st day	2nd through 6th day	2nd week through 1st month	2nd through 6th month	7th through 12th month	1 to 2 yr. after	2 to 3 yr. after	3 to 5 yr. after	5 to 10 yr. after	10 to 20 yr. after	Over 20 yr. after	Unknown	Totals
Cervical	Service...	0	0	0	0	3	0	2	2	1	0	0	0	0	8
	Civilian ..	83	31	16	9	6	4	2	0	0	0	0	0	2	153
Thoracic and Lumbo-sacral	Service...	0	0	0	0	21	4	17	14	2	1	0	0	1	60
	Civilian ..	37	5	10	10	7	4	8	2	6	0	1	2	1	93
Caudal	Service...	0	0	0	0	7	3	12	3	0	0	0	0	1	26
	Civilian ..	14	1	3	4	3	5	1	2	1	6	2	1	0	43
Epidural Abscess, etc.	Service...	0	0	0	0	0	0	0	0	0	0	0	0	0	0
	Civilian ..	0	2	4	7	3	0	2	0	0	2	1	0	0	21
Totals	Service...	0	0	0	0	31	7	31	19	3	1	0	0	2	94
	Civilian ..	134	39	33	30	19	13	13	4	7	8	4	3	3	310

Bed Sores. Perhaps the most important and certainly the most frequent reason for prolonged hospitalization of the so-called "paraplegic" veterans is the bed or pressure sore. Fifty-three per cent of the en-

tire group of 212 patients had or have open bed sores that required active therapy. Forty-one per cent of the patients still in the hospital are there in part or wholly because of their bed sores, while 69 per cent of the patients that have been discharged had bed sores treated during their stay, 31 per cent being multiple. Many of the others had scars of sores that had healed before their arrival at Cushing. In comparison, it should be noted that only eighty-nine or 28 per cent of the 313 civilian cases had bed sores. This includes all cases regardless of length of stay in the hospital and so may be slightly biased toward the low side. If only the 204 patients that were discharged alive from the hospital are considered, the number having bed sores is reduced to sixty but the incidence remains the same— 29 per cent. If the twenty-four patients that died during the first twenty-four hours after injury are eliminated as not having lived long enough to develop sores, the incidence in the remaining 289 was 31 per cent, a figure less than one-half of the discharged service group. Emphasis has been placed upon this comparison because in ideally handled cord and cauda equina injuries, bed sores should never occur. This was pointed out in 1940[5,6] and further experience merely confirms this statement.

The causes of bed sores are two: failure to restrict weight-bearing on any given bony prominence to not longer than two hours at a time and failure to maintain the patient in a constantly dry bed. Once bed sores are started they are incurable in the presence of involuntary muscular spasms, hypoproteinemia, malnutrition and anemia. The most prolific source of trouble and a virtually certain cause of bed sores in the "paraplegic" is plaster of paris dressings and of all others, plaster of paris body casts are the most harmful. With a body cast in place it is impossible to move the patient and relieve the local pressure its presence produces. Moving the patient and the cast does no good. I have been unable to determine the number of "paraplegic" soldiers that were put into plaster of paris

jackets preparatory to transport to this country from overseas but certainly the figure is large. This, like the universal use of suprapubic bladder-drainage, was considered to be justified by reason of the uncertainties of transportation and because of

TABLE VI
TYPE OF BONY INJURY

	All Cases	Transection	Partial Cord Injuries	Cauda Equina Injuries	Radiculitis Only
		No. of Cases of			
Major { Service	43	19	13	11	0
Major { Civilian	214	51	128	28	7
Minor { Service	12	8	2	2	0
Minor { Civilian	16	3	10	1	2
None { Service	19	9	5	5	0
None { Civilian	76	19	41	14	2
Total of { Service	74	36	20	18	0
Above { Civilian	306	73	179	43	11
No Note { Service	26	11	5	10	0
No Note { Civilian	7	4	2	1	0
Totals { Service	100	47	25	28	0
Totals { Civilian	313	77	181	44	11

the difficulty of ascertaining the extent and need for protection of the bony injury in any single instance. No criticism can be made of this decision but it is profitable to consider what it leads to and whether in the future it can be altered for the greater ultimate benefit of similar patients. That this has its practical aspect is evident from the comparative figures cited above. The low incidence of bed sores in the civilian group is attributable to the fact that plaster of paris was never used and because for the last several years turning every two hours, dietary measures and prevention of maceration of the skin of the back by proper nursing and the use of tidal drainage have all been rigorously adhered to.

The use of plaster of paris casts is justified, *if at all*, by the necessity of splinting the injury to the vertebral column. My experience leads me to believe that this justification is present only under combat conditions, and that in the absence of such

conditions casts are unnecessary. A comparative analysis of the appropriate figures in these two series of patients supports this contention. The relative occurrence rate of a significant bony injury is 75 per cent in the civilian and 74 per cent in the service group, the significant difference being found in the severity of the injury. In the civilian group 70 per cent had major and 5 per cent minor bony injuries, while in the service group only 57 per cent had major and 17 per cent minor bone injuries. (Table VI.) It is apparent (Table VII) that not only were bed sores more than twice as frequent in association with bone injuries in the service groups but were more than two and one-half times as frequent in the absence of any bone injury whatsoever. Since the therapy has been virtually the same in both series, once the patient reached his final hospitalization, this significant difference and the resultant delay in rehabilitation must be ascribed to the methods used in the services for treatment prior to that time, and speaks strongly against the use of plaster of paris or other similar methods of immobilization in patients with spinal cord and cauda equina injuries.

Genitourinary Tract Disease and Injury: Renal and Bladder Stones. The second most disabling complication is disease and injury to the genitourinary tract. It is impossible to deal with all phases of this major problem but certain aspects may be briefly touched upon. The occurrence of renal and bladder calculi has been extremely frequent among the "paraplegics" in the services. Many papers have been written on the subject[7-12] and there can be no doubt but that it has constituted a great hindrance to the rehabilitation and sometimes to the saving of the life of these patients. In this group of 100 patients, fifteen had bladder and fourteen kidney or ureteral stones, an incidence of 29 per cent. In the civilian group there were eight bladder stones and three renal stones, an incidence of 4 per cent if only the 252 patients that lived eight days or longer after injury, that is, long enough to develop stones theoretically, are considered. Five of the eleven patients that had stones had long histories of treatment elsewhere with formation of stones during that time, thus reducing the actual incidence of stones under treatment by tidal drainage to six in 252 patients, or 2 per cent. This is too great a discrepancy to be passed over lightly especially when the virtual parallellism of the two series is taken into consideration. Special diets,[13] elevation of the head, etc.,[8,12] have been proposed as being necessary for the elimination of genitourinary tract calculi. I can only state that no such measures were even considered, much less carried out, in this civilian group of cases. The most important difference between the two series of cases, however, is, as pointed out, one of the time of application of what I have come to regard as the essential minimal therapy of the genitourinary tract in cord injury patients. This requires, among other things, the use of tidal drainage with its maintenance of normal anatomic and functional states of the bladder and its prevention of major infection of the genitourinary tract.[14] A constantly dry bed and the ingestion of adequate amounts of water are also essential.

Practically all of these requirements were unfortunately disregarded or omitted of necessity during the first stages of treatment of the service "paraplegics." Most

TABLE VII
RELATIONSHIP OF BED SORE TO BONE INJURY

	Bed Sores Removed with a					
	Major Bone Injury		Minor Bone Injury		No Bone Injury	
	No.	Percentage	No.	Percentage	No.	Percentage
Service Cases..........	29	41	9	13	13	19
Civilian Cases.........	62	20	3	1	20	7

had and all were supposed to have had a suprapubic bladder fistula produced. This violates the anatomic and functional normalcy of the bladder. In addition, after the suprapubic fistula was closed, tidal drainage was often not used for a considerable period of time. As a result the bladder either shrank and became overactive or stretched and become virtually atonic. Infection involving both the bladder and kidneys was inevitable under such conditions. Because the infection was subclinical did not mean it was any the less lethal in so far as serving as a groundwork in which stones could and would form.[15] To illustrate the importance of this there were twenty-five major infectious processes affecting the kidneys, ureters and bladder in the service and only nine in the civilian group. The comparative percentages are 25 and 4. It seems reasonable to assume in the light of these figures that the presence of infection is a major and may be the determining factor in the production of renal and bladder stones in "paraplegics." The custom of draining their bladders by a suprapubic fistula certainly does not detract from this assumption. Albright et al.[15] and Albright in a personal communication states that among other causes of genitourinary tract calculi common and important ones are the development of a nidus without local stasis at any point of injury to the mucous membrane, the persistence of an alkaline urine and tubular damage without associated glomerular damage. This last leads to hypercalcuria and is caused by chronic genitourinary tract infection which is usually staphylococcal and often subclinical. It would appear then that there is greater chance of preventing the formation of renal calculi in "paraplegics" by concentrating on prevention and control of genitourinary tract infection and subsidiary conditions such as an alkaline urine than by manipulation of diets and the use of other less direct procedures. Experience has demonstrated that the prompt and continued use of tidal drainage[16] is the best method of doing this.

Resection of the Internal Urethral Sphincter. Emmett[17] and Thompson[18] have advocated resection of the internal urethral sphincter in patients with cord injuries, tabes dorsalis and a theoretic congenital thickening of the internal sphincter that causes so-called "unexplained cord bladder." It is clear that they have practiced this operation only on patients that have had their genitourinary tract lesions for a long time. It is also clear that their experience has not included patients with cord injuries during their earlier stages and they are therefore not familiar with the stages of recovery that a bladder passes through if properly treated. By implication and omission they would lead the careless reader to conclude that resection of the internal urethral sphinter is appropriate and therapeutic in all patients with cord injury regardless of the length of time that has elapsed since injury and regardless of the treatment they have received for their bladder. There is also a widespread belief among surgeons and patients to this same effect and some tendency to regard this operation as a panacea in the care of the genitourinary problems in "paraplegics." The operation is undoubtedly essential in the demonstrated presence of hypertrophy or other similar affection of the internal sphincter if the latter prevents evacuation of the bladder at every emptying contraction. The operation is not without danger, however, in that if too much tissue is removed the patient becomes permanently incontinent, a condition which cannot then be controlled even by an inlying catheter. The procedure also appears to be unduly risky and probably unsuitable for women because of the peculiar anatomic distribution of the sphincteric muscle fibers. It is contraindicated in the face of a flaccid or spastic external sphincter.

I believe that the need for this resection can virtually be eliminated if proper care is provided for the bladder from the start. A comparison of the appropriate data in both of the present series of cases will bear this

out. Nine of the service patients had their internal sphincters resected. Six had transected cords and three cauda equina injuries. Four of the transected cord cases had "spastic" and two hypertrophied internal sphincters. In the cauda group one resection was done on a spastic and the other two on hypertrophied sphincters. There were four other patients with spastic sphincters that were not operated upon. In the civilian group only two patients needed and received resection. They were both patients that had had long periods of poor bladder therapy elsewhere before coming under my care and were both transected cord cases.

The only essential difference in these two series is found in the immediate and early method of dealing with the bladder. None of the patients treated from the start with tidal drainage and hence not subjected to prolonged periods of stretching, contraction or infection of this organ gave any evidence of any sphincteric condition that would necessitate resection. On the other hand, eleven patients whose bladders had not been similarly treated but had rather been subjected to a variety of physiologic insults did require resection of their internal sphincters before the organ could empty itself. To be sure, except for one patient who developed an incurable incontinence after the operation, all had a satisfactory postoperative result but this is certainly not enough to justify indiscriminate use of the procedure in "paraplegics" especially in the light of the success of the non-operative methods used in the larger series. Finally, in consideration of the incidence of the conditions calling for resection in the smaller series it is obvious that under similar conditions, if the operation has the widespread need that has been suggested, an analogous series three times as large should be expected to produce as many or more patients needing operation. The fact is that no resections were either indicated or necessary. The operation of resection *per se* will not be needed, therefore, to assure emptying of an "automatic," reflex

or controlled bladder and if it is needed, is evidence of improper earlier therapy.

Flaccid Sphincters. Certain patients in both series had flaccid or completely atonic urethral sphincters. There were twelve such cases in the service and one in the civilian group. All occurred in patients with cauda equina injuries either alone eleven or in combination with an injury to the lumbosacral cord two. All showed evidence of denervation, both sensory and motor, of the peripheral field supplied by the sacral nerves. By cystometrogram four had atonic, eight autonomous and only one a reflex bladder. The conclusion is inescapable that the sphincters and all but one of the bladders had been denervated as the result of the central nervous system injury. As far as I know or can find out, this condition has never before been either described or recognized in connection with cauda equina or low spinal cord injuries. The constant wearing of a urinal is the only present method of dealing with the incontinence that goes with it.

Spasm. The third major impediment to the rehabilitation of patients paralyzed as the result of spinal cord and cauda equina injuries is "spasm." The most disabling variety of spasm is the involuntary variety, usually but not necessarily adductor-flexor in type, which involves not only the lower extremity but the lower back and abdominal muscles as well. In experimental animals, this is a constant accompaniment of an anatomic transection of the spinal cord at any level above the conus, and has been described under the term "mass reflex." It has been said not to develop in association with non-transecting cord injuries.[16] In any event it does not put in its appearance until spinal shock has worn off. In man the situation, although described as being the same as in animals, appears in the light of greater experience to be somewhat different. Involuntary spasm or the "mass reflex" is not, for example, a constant accompaniment of transection nor is its absence pathognomonic of a non-transecting injury. If all the transected and

partial cord injuries in the two groups are combined and only those patients are included that have lived three months or longer and thus have had an opportunity to develop spasms, there are ninety-one transections and 148 partial cord injuries himself and his patient to any such destructive operation as anterior rhizotomy in his attempts to relieve this disabling condition.[1] This is more true because this may be the only way to control the spasms and make rehabilitation possible. In patients

TABLE VIII

INCIDENCE OF INVOLUNTARY SPASM

	Died within 3 mo. of Injury			Total Cases			Cases That Could Have Developed Spasm			Severe Spasm			Mild Spasm			No Spasm			No Note		
	Service	Civilian	Total	Service	Civilian	Total	Service	Civilian	Total	Service	Civilian	Total	Service	Civilian	Total	Service	Civilian	Total	Service	Civilian	Total
Transection of Cord.........	0	33	33	47	77	124	47	44	91	16	21	37	13	0	13	17	21	38	1	0	1
Partial Cord Injury.........	0	58	58	25	181	206	25	123	148	11	3	14	2	1	3	12	119	131	0	0	0
Cauda Equina Injury........	0	0	0	28	44	72	0	0	0	0	0	0	0	0	0	28	44	72	0	0	0
Totals.................			91			402			230			51			16			241			2

to be considered. (Table VIII.) Of the transections—and that means loss of all sensory and voluntary motor function below the level of the cord injury—50 or 55 per cent had spasms. Of these only thirty-seven or 41 per cent had severe enough spasms to justify therapy. In the light of these figures it is impossible to contend any longer that involuntary spasms or the "mass reflex" is a necessary accompaniment of transection of the spinal cord in man. Of the 148 partial cord injuries seventeen or 11 per cent had spasms, fourteen or 9 per cent being severe enough to prevent ambulation completely. It is evident that invaliding involuntary spasms that are indistinguishable from those described as constituting a "mass reflex" *do* occur in patients who retain sensation and a varying degree of voluntary motion below the level of a partial cord injury.

These considerations are of great importance because the responsibility rests squarely on the surgeon's shoulders to determine by every means in his power the degree and type of injury that has been done to the spinal cord before committing with transected cords the usual root section can be justifiably carried out. If the cord lesion is known to be only a partial one, however, the rhizotomy must be done in such a way as to preserve the already existing voluntary motion. I believe that this important differentiation can be met only by actual operative visualization of the injured areas. Moreover, this should be supplemented by electrical stimulation of the cord above and below that area before a rhizotomy is justified. On that basis, and with the above requirements met in every case, only twenty-seven patients or 21 per cent have had rhizotomies out of this group of eighty transections and forty-eight partial lesions. Four additional service patients would not allow the operation to be performed and twelve were still to be done in October, 1947, on service patients that were still hospitalized. On the other hand, fifty-four exploratory laminectomies have been advised and fifty-three performed on the seventy-two discharged service patients in the transection and partial cord injury groups. Forty were done on the transected and thirteen on· the partial cord injury

cases. I am convinced that this realistic approach to the treatment of spasms has materially hastened and greatly improved the rehabilitation program and, at the same time, has avoided doing harm to and depriving any patient of any useful neuromuscular activity he would otherwise have had.

RESULTS OF TREATMENT

In measuring the success or failure of any program directed toward rehabilitating "paraplegics" there are many other factors to be considered beside the patient's ability to ambulate. Failure to itemize *all* of these factors casts doubt upon the claims of the sponsor.

Ambulation. It is not enough to know that a patient can get about; one must also know *how much* he can get about. We do not consider that a patient has satisfactorily finished his ambulation training until he not only can but prefers to wear his braces all day, is able to walk a thousand yards without stopping, to go up and down all stairs no matter whether they have a hand rail or not, to go up and down curbs freely, to get in and out of all types of chairs, to get himself off the ground without help if he falls, to walk in city traffic unaided and safely, to use all public conveyances without help, to live in his original home without demanding significant alterations, to use the toilet and bath-tub as usual and to live as normal a social and work life as the enforced use of braces and crutches will permit. We have met these self-imposed requirements in sixty-two or 62 per cent of the service patients that form the subject of this report. Our greatest success came in the group with cauda equina injuries. All but three of these twenty-eight patients or 98 per cent acquired all the skills listed above as well as some others. Twenty-three or 49 per cent of the patients with transected cords and fourteen or 56 per cent of those with partial cord injuries did the same thing. A less satisfactory but workable rehabilitation satisfied another group. They are able to walk only 500 yards, do not wear their braces as long, use only stairs with rails, have to be helped into public conveyances, and in general are less active and skilled. As a result certain alterations have had to be made in their homes. This and the preceding group were able to obtain and learn to drive an automobile. They usually were licensed on our recommendation by the state authorities. The combination of available transportation of their own, adequate social activity, plus an inability or disinclination to perceive the benefits that would accrue to them by finishing the full course of ambulatory training proved too much for seventeen such patients. They were discharged with our blessing but without our approval, the more so as there was no way by which they could be detained. It was hoped that perhaps, some of them would return later when they had learned by practical experience how much they were missing. Eleven came from the transection, four from the partial and two from the cauda equina group of injured. Thus seventy-nine or four-fifths of 100 "paraplegics" have been discharged during the past year able to ambulate enough to live at least a restricted social and work life.

Twenty-one did not attain ambulatory ability commensurate with the above. Two of these were discharged dead without ever getting out of bed and while still in the hospital, seven left in wheel-chairs and twelve were satisfied with a swing-through gait but no more. These last twelve were the only patients of the twenty-one who might have made something more of their opportunities but did not do so. The reasons follow: one still remains A.W.O.L., four were discharged for disciplinary reasons and seven left against medical advice.

All but one of the seven that were discharged in wheel-chairs and without having had any ambulation training were irreclaimable at the time they left. Three of them refused even to permit measurements to be taken for braces stating that they had no intention of ever learning to ambulate. One also had disabling

spasms which could be corrected only by surgery but would not permit this to be done. Two of the remaining three were limited to wheel-chairs by a floating femur with osteomyelitis of the head, neck and acetabulum, and by quadriplegia complicated by a paranoid schizophrenic personality, respectively. The remaining patient was transferred at his own request to another Veterans' Administration Hospital for his ambulation training.

As far as ambulation goes our program was a failure in twenty-one of the 100 patients. This was ascribable to the untreatable nature of the disease in five; to the patient's own peculiarities in nine and to the doctor's inability to hold the patient's interest in seven.

Control of the Bladder. As important to the patient from the point of view of a decent social and work life as ambulation is his ability to control his bladder and bowel. Because it can be attained if the patient cooperates and the doctor makes use of the knowledge available to him, I believe that anything short of perfection in these two functions is unjustifiable except where there is actual anatomic deficiency. Thus a patient is only considered to have obtained maximum benefit from the therapy he has received when he is able to go through a normally active day without getting wet and can regularly sleep through the night without having to get up and without getting wet. The wearing of a urinal or other apparatus is not countenanced unless the patient has loss of control by reason of irreversible anatomic changes. If his internal and external sphincters are atonic or his bladder has been allowed to stretch to a point where its tonicity is permanently destroyed or to shrink to a point where its destensibility is gone and its capacity reduced to only 2 or 3 ounces or less, then and only then is the wearing of some form of urinal permitted. The only exception that is made is for the patient who has to take a long trip on which he will be unable to empty his bladder at the regular intervals that he is accustomed

to. Then a urinal is considered justifiable as insurance against wetting. In particular, we feel very strongly that an "automatic" bladder need not and should not be considered a satisfactory end point by either the doctor or the patient.

An analysis of the urination situation in the 100 discharged patients demonstrates that we have attained our self-imposed goal. Forty-five patients had acquired twenty-four-hour control by the time they left the hospital. Nineteen had day control only. They did not have night control because they would not adjust their water intake.[16] If they had been willing to do this, they could have had twenty-four-hour control. Forty-four of the sixty-four that had either twenty-four-hour or day-control used no urinal at any time. The other twenty as well as eight patients who had no control used a urinal on trips only. There was no information as to use of urinal by ten patients. Thus forty-five of ninety-eight, (46 per cent) had full control and sixty-four or 66 per cent had day control. Forty-four of ninety or 49 per cent did not use a urinal at all and twenty-eight or 31 per cent used a urinal on trips only.

Thirty-six patients were discharged without bladder control. Twenty-four or 27 per cent used a urinal at all times and four or 11 per cent used tidal drainage at home. In fifteen the lack of control was the patient's own fault. They usually preferred not to take the trouble to either learn bladder training[16] or to regulate their water intake and in consequence not only had no control, but also had to use a urinal constantly. In three the lack of control was inherent in the patient's condition (two deaths, one quadriplegic patient), in ten it was inherent in the bladder and in seven in the sphincters either alone or in conjunction with the bladder condition. The cause of this latter difficulty was the denervation of the organ and its musculature by the cord or cauda equina injury with the production of either atonic or autonomous bladders and flaccid sphincters. In only one patient was the lack of control unexplainable and hence charge-

able to the doctor. Thus it has been possible to teach all except one of the patients that were physically capable of learning to do so and that were cooperative and willing to take enough time and trouble, to have twenty-four-hour control of their bladders without the use of a urinal at any time except as a safeguard on long trips when the bladder could not be emptied at the required intervals.

Bowel Control. It is only slightly less important for these patients to be able to control their bowel. Here again we believe that we are derelict in our duty if anything short of twenty-four-hour control without leakage or soiling, the use of enemas, digital emptying or any similar procedure and with only minimum use of a bland cathartic is countenanced as a satisfactory end result. We were able to discharge seventy-one of ninety-three patients as having met these requirements. Information as to the bowel condition on discharge is not available in seven. Only six were taking enemas regularly at discharge.

Sixteen patients were discharged without bowel control. In nine this was the patients' fault in that they were discharged for disciplinary reasons two, were A.W.O.L. two or were uncooperative five. Six had no control because of their physical condition, one having died and five being afflicted with atonic sphincters because of denervation as the result of the cord or cauda equina injury. One other left to go to another Veterans' Administration hospital for completion of his treatment. Thus, there were no patients that lacked bowel control on account of the doctor's failure.

Bed Sores. As noted above, 71 per cent or seventy of the ninety-five discharged patients about whom the information is available, had bed sores that required treatment during their stay. In ten the bed sores were still present on discharge. Four were given medical discharges despite that fact. The decubiti were small and were located in the gluteal fold, on the sacrum, over the trochanter and on one heel, respectively. One patient died in the hospital,

two were discharged for disciplinary reasons, two left against advice and one was transferred to another Veterans' Administration hospital at his request. The scars in nineteen others were adherent but they were either so placed or so small that it was not anticipated that they would break down. Twenty-six had non-adherent and fourteen padded scars. In fourteen there had been no note made about the condition of the scar. Thus, only four patients out of fifty-six that had had bed sores and about whom a note was made relative to their condition at discharge can be said to have been allowed to leave the hospital before a proper end result had been obtained.

Pain. Pain did not prove to be the major problem that was expected. Sixty-five of the ninety-nine patients about whom this information was available had no pain on discharge. Another twenty-seven had minor degrees of pain that were no more than bothersome. This type of pain was distributed about equally among the transected (ten), the partial cord injuries (nine), and the cauda equina cases (eight). Active treatment was neither indicated nor necessary in any of these cases. Six cases had major disabling pain on discharge. All were in the cauda equina group of injuries. One patient in this last group died from the infection which caused his pain. Another complained of severe pain in his rectum the cause of which was unknown and which was not relieved at the time of his discharge against medical advice. The available records gave no information about his treatment. Of the remaining four, three had a radicular type of pain. Two of the three had posterior rhizotomies and one a cordotomy in addition without relief. The fourth left without having received any active therapy and after having completed his ambulation training, but with an autonomous bladder, spastic urethral sphincters and no urinary control. His bowel control was satisfactory. This last case had severe leg pain of an atypical type. He was a constitutional psychopath, would not eat properly and was frequently under the

influence of alcohol. He refused all treatment except drug therapy with opiates and barbiturates and was discharged against medical advice.

Spasms. Spasms may be enough to prevent rehabilitation even though all other disabling conditions have been cared for. It is therefore essential to know about their presence in the discharged patients. Such data are available in ninety-eight patients. Sixty-two were discharged without spasms and twenty-two with minimal spasms that caused no interference with ambulation or bladder and bowel control. Fourteen had spasms that were bothersome or worse at discharge. Ten of these had had a transection and four a partial injury of the cord. Only three cauda equina cases had spasm of any type. It was minimal and in every instance there was an associated injury to the lower end of the cord in addition to the damage to the cauda equina.

Three of the fourteen patients with severe spasms were disabled by them. One of these refused an anterior dorsolumbar rhizotomy and became an able ambulator nevertheless; one had an ineffective anterior rhizotomy and was discharged against medical advice while still in a wheel-chair and the other was a schizophrenic quadriplegic who was discharged in the same way and in the same condition. In nine the spasms were only bothersome. Two of these were discharged for disciplinary reasons— one after refusing rhizotomy—but not on that account; four were discharged against medical advice one having refused anterior rhizotomy. None of these six were able to ambulate beyond the first stages at the time of their discharge. One was sent to Cushing Veterans' Administration Hospital having just graduated from a wheel-chair existence. The other two were able to ambulate fully. They were given medical discharges despite the presence of spasm. One patient had minimal spasms. He refused an anterior rhizotomy and was given a disciplinary discharge for other reasons. The last was given a disciplinary discharge while still in a wheel-chair, the degree of

his spasms at that time not having been noted. Thus the presence of significant spasms at discharge may be considered the patients' fault in eleven and the doctor's fault in two. In connection with this it should be noted that only thirteen rhizotomies were performed and only seventeen recommended in this group of 100 patients, the other four having been refused by the patients. Of the thirteen that were carried out, ten were anterior dorsolumbar and three posterior in type. One patient was relieved of disabling typical abdominal flexor spasms associated with a mid-thoracic transection of the cord by injection with novocaine of the appropriate peripheral nerves at their exits from the intervertebral foramina.

Sexual Status. It is not possible to give any figures on the sexual status of the patients in this series. An investigation started one year ago in an endeavor to bring some order out of the chaos of irresponsible statements that are in the medical literature has demonstrated that the problem .is so complex that much more work and time must be spent on it before any conclusions that even approach reliability can be drawn.

Patient's Attitude toward the Treatment He Has Received. In addition to the physical one this group of injured has a tremendous psychologic problem to face. We believe that we can to some degree, measure the success of our treatment of the latter by delving into the psychologic field far enough to attempt to determine the patient's own reaction to this therapy. These patients, therfore, were all asked at discharge whether or not they had been satisfied with the treatment they received while under the Veterans' Administration care at Cushing General Hospital. Answers were obtained in ninety-eight of the 100. Seventy-nine expressed themselves as satisfied, eight as not satisfied and five asked to be transferred to other Veterans' Administration Hospitals. Five were given their discharges for disciplinary reasons, fourteen

left against medical advice, three were A.W.O.L. and two died in the hospital.

Of the eight that left unsatisfied with the treatment, six had transected cords and two partial cord injuries. One of the transected patients left after eight operations which all originated with the implantation by some surgeon overseas of a Lane's plate in a dirty spinal wound. Four were obviously emotionally upset, one having his judgment warped by his poor general condition, another because attempts were made to force him to ambulate against his will, a third because "everything was wrong" and the fourth as the termination of a consistently and continuously recalcitrant attitude. The sixth left without giving a reason. The first of the two partial cases was a schizophrenic quadriplegic and the second left following and probably because of an unsuccessful anterior rhizotomy.

Discipline has not presented a major problem. I consider that this is traceable to two reasons: First, all patients are given a schedule which keeps them occupied in some activity looking toward their more rapid discharge and more complete rehabilitation and, second, because the attitude of the staff is that the patients are in the hospital only temporarily and as a means to an end. Repeated infractions of the work schedule, excessive use of alcohol, foul or obscene language and major insubordination are followed by a hearing of all parties before a board and the discharge of the patient for disciplinary reasons if the charges are approved by the board. This procedure is adopted as a last resort only. The local paraplegic association has proved extremely helpful in maintaining discipline once its members understood that cooperation with the medical program meant quicker and more complete rehabilitation. Only five patients were discharged for disciplinary reasons during the year. Two were because of the persistent use of alcohol; one because of being persistently A.W.O.L.; one because he refused to take any part in the medical program and the last because of driving an automobile on the public highways before he was able to do so, without our permission and without being licensed by the state.

Training for and Provision of Employment after Discharge from the Hospital. The most difficult, the most important and probably the least well handled of the whole problem of rehabilitating the spinal paralytic is to provide him while in the hospital not only with the potential ability to earn his own living but with employment after he leaves the hospital. No matter how medically fit these patients are on discharge, if the mechanism is not provided for them whereby they can earn their own living in their community and thus maintain their self-respect after leaving the hospital, all the professional care that has been lavished on them will be wasted. It is unreasonable to expect the average family or friend of a "paraplegic" to assume the same detached attitude toward him that they would toward a normally equipped wage-earner. They cannot and will not make him work even if he is able to do so and will have no part in forcing him to train himself for work if he has not already acquired that ability. Indeed, the weight of community public opinion as well as the emotional response of his family will make it extremely difficult if not impossible for the "paraplegic" who wants to work in spite of his handicap to do so. His family feels guilty if they do not see that he need carry no responsibility in the home. The community through the various charitable organizations takes the same attitude. Employers, in the main, regard the employment of a "paraplegic" not in the light of buying competent labor but rather as a noble gesture or as "part of their war effort" and lacerate the "paraplegic's" pride and self-respect by paying little or no attention to his skill or his worth as a member of their organization. Only prolonged education of the employing and employable population and of the parents and friends of these patients can correct this. Rather than force or allow the "paraplegic" to combat this attitude unprepared

he must be given the opportunity to learn a trade that is within his physical capabilities while in the hospital, and gainful employment in that trade must be waiting for him on his discharge. We have failed badly in meeting these responsibilities. The reasons are many and complicated but the reason that most people will assume as the chief one is not a major factor. This is the unwillingness of these patients to work. Only six out of the 100 that were discharged made the statement that "they did not intend to work but were going to let the government take care of them" for the rest of their lives. This is certainly no more than the number of employables with that attitude in the community at large. It is only surprising that it is not larger. These patients, being totally disabled, will get paid at least $360 a month by the Federal government and in certain states will receive additional sums that bring the figure close to $500 a month for the rest of their lives. This is sufficient for almost anyone to live on comfortably without further income and it is a most effective deterrant to starting on a new, strange job. On the other hand, it is less than these men deserve from the country and any effort looking toward its reduction is unthinkable. We should, however, see to it that they are in a position to use this money to their own best advantage. This means gainful labor in the case of a male adult.

We have information on fifty-five potential wage earners relative to their capacity and willingness to do gainful labor on discharge from the hospital. Thirty of this group had a job at gainful labor or its equivalent in educational training. Twenty-five did not. The forty-five other patients fell into a different category. It was physically impossible for three to hold a job on discharge. Seventeen separated themselves from the hospital by their own actions before arrangements for their training and placement could be completed; two being A.W.O.L. constantly, seven having left against medical advice, five having been discharged for disciplinary reasons and three having gone to other Veterans' Administration hospitals. There was no information about employability or employment in the records of twenty-five patients.

It seems to me that despite the quality of medical care, which the figures show to have been good, and in spite of the fact that unwillingness on the part of the veteran to work played no part in the picture, we have fallen far short of the necessary requirements for rehabilitating these patients. Our shortcoming lies in failing to see to it that they are trained in wage-earning skills during their hospital stay and that they are provided with gainful labor in accordance with this training on their discharge to civilian life again.

SUMMARY AND CONCLUSIONS

1. The results of the treatment of 212 veterans suffering from wounds and injuries of the spinal cord and cauda equina —including "paraplegics"—at Cushing Veterans Administration General Hospital during the year from October 1, 1946 to October 1, 1947, is reported.

2. One hundred patients were discharged and 112 remained for further treatment. There were forty-three readmissions.

3. Seventy-nine patients were fully ambulatory at the time of discharge.

4. Of the twenty-one that were not ambulatory at discharge five were failures because of the untreatable nature of their disease, nine because of the patients' own peculiarities and seven because the doctor failed to hold their interest.

5. Sixty-four of the 100 patients were capable of acquiring full control of urination: forty-five had twenty-four-hour control, nineteen had day control only, forty-four used no urinal at any time and twenty-eight used a urinal on trips only.

6. Thirty-six patients were discharged with no control of urination. In fifteen the lack of control was the patients' own fault, in three it was inherent in the patients' condition, in ten it was inherent in the condition of the bladder or sphincters or both and in one was unexplainable.

7. Seventy-one patients were discharged with full twenty-four-hour bowel control.

8. Pain was not a major problem. Only six patients (all cauda equina injuries) had enough pain at discharge to warrant treatment.

9. Disabling spasms were present in fourteen patients at discharge. Sixty-two patients were discharged without spasm and twenty-two with minimal spasms that caused no interference with the patients' ambulation or bodily functions.

10. All spasms when present were associated with a cord and never with a cauda equina injury.

11. Thirteen rhizotomies were performed, ten anterior dorsolumbar and three posterior in type.

12. Seventy-one patients had bed sores that required treatment during their stay. Bed sores were still present in ten at discharge.

13. Discipline was not a problem, only five of the patients having been discharged for disciplinary reasons.

14. Much still needs to be done in educating the public, these patients and their families as to the necessity of training these paralyzed veterans for gainful labor during their hospital stay and providing them with gainful employment and the ability to be self-supporting when they leave the hospital.

15. Evidence is presented by means of appropriate comparison with a civilian series of 313 patients with spinal cord and cauda equina injuries to demonstrate that: (1) The use of plaster of paris body casts is conducive to the formation of bed sores; (2) renal and bladder stones can be virtually completely prevented by the early persistent and proper use of tidal drainage alone; (3) need for transurethral resection of the internal urethral sphincter to restore micturition is evidence of improper earlier care of the bladder and is not otherwise obligatory in these patients and (4) the spasms characterized by the term "mass reflex" occur in both complete transections and partial injuries to the spinal cord in man.

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ACUTE ABDOMINAL EMERGENCIES IN PARAPLEGICS*

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DURING the past five years medical literature has stressed, time and time again, the fact that the postwar years would bring diagnostic problems to the civilian physician which he had not previously encountered.[1,2] The profession at large has been cautioned, even in apparently simple diagnostic problems, to bear in mind the possibility of recurrent malaria and various other exotic diseases when dealing with returned overseas veterans. In addition to the tropical diseases to which the returned military may be heir, there are other formerly rare maladies which the war has made common place. It was estimated that at the close of hostilities there were approximately 2,000 paraplegic patients in army, navy and veteran administration hospitals.[3] Because of the progress made during the war years in the management of these patients, the majority will be rehabilitated to the point of assuming a happy and useful life in the civil community. As this group of paraplegics returns to civil life they will bring to the civilian physician many diagnostic and therapeutic challenges.

One of the most difficult problems which the paraplegic patient presents is that of the acute abdominal emergency. Because of the extreme debilitated state of such patients when returned from overseas areas of combat, because of the foci of infection in the decubitus ulcers and genitourinary tract, because of the tendency of these patients to genitourinary calculus formation and because of their low resistance to intercurrent disease, acute abdominal emergencies were seen far more frequently in a group of patients in a paraplegic center than in an equal number of healthy young adults. It is believed that these patients after leaving these centers will continue to present perplexing syndromes of abdominal distress to the civil physician. The presence of a profound neurologic lesion, which usually results in anesthesia below the level of injury, makes the evaluation of these episodes an extremely difficult matter. The purpose of this paper is to enumerate certain factors which may be of assistance in evaluating symptoms of abdominal distress in the paraplegic patient.

MECHANISM OF VISCERAL PAIN CONDUCTION IN COMPLETE SPINAL CORD TRANSECTION

It has been definitely established that the abdominal viscera are supplied with sensory nerve endings, and that painful sensation may be conducted from the viscera to the spinal cord by way of the thoracic, splanchnic, hypogastric and pelvic nerves and thence over the posterior nerve roots.[4] White has pointed out that even though these visceral sensory neurones resemble sympathetic motor neurones, they are anatomically as well as physiologically different. He states further that it is incorrect to refer to sympathetic pain fibers, but best to think of the visceral innervation as composed of mixed nerves, containing a large preponderance of sympathetic motor fibers with a small admixture of sensory fibers which belong to the posterior spinal root.[5] Nevertheless, despite the fact that visceral sensation is conducted by the posterior root system in a similar manner to the pain conduction from the abdominal wall or from the extremities, there must be a dissimilarity between the pathways of reception of

visceral sensory impulses and somatic sensory impulses. Many paraplegics with a physiologically complete transection of the spinal cord cannot perceive any sensory stimulation of the somatic type below the level of spinal cord transection, but can perceive painful stimulation of the viscera whose sensory fibers are believed to enter posterior roots below the level of transection or compression of the cord.

We have seen several paraplegic patients with abdominal pain who were known, by means of exploratory laminectomy, to have complete spinal cord transection, but who, nevertheless, developed abdominal pain from disease of viscera whose sensory nerve fibers are reputed to enter roots below the level of transection. Livingston states that it is particularly notable that visceral pain which is satisfactorily relieved by bilateral chordotomy often gradually returns, and that visceral pain pathways may have accessory pathways by which they reach the sensorium.[6] Karpus and Kreidl suggest that such accessory pathways may be represented by relays of short neurons close to the spinal gray matter,[7] and the escape of such pathways from damage might account for the presence of visceral sensation in the absence of all somatic sensory perception in a rare case. Foerster has suggested that these visceral impulses of pain may pass upward for several segments in the sympathetic ganglionic chain before entering the cord.[8] This is believed to be the explanation in several of our own cases.

One case cited below, however, cannot be satisfactorily explained by any of the above postulations. This young male was rendered quadriplegic following fracture of the fifth cervical vertebra. Exploratory laminectomy revealed a complete transection of the spinal cord in the region of the sixth cervical nerve root. Nevertheless, this patient developed sharp stabbing pain in the epigastrium due to a gastric ulcer, the presence of which was confirmed by x-ray. In this case it is impossible to assume that the afferent pain impulses were transmitted cephalad either by accessory pathways in the cord or by fibers running in the sympathetic chain to enter the cord above the site of the transection. The vagus nerve has not yet been demonstrated to carry painful impulses *per se* from the abdominal viscera.[9,10] White states that while the vagi carry some afferent reflex stimuli and a part of the sensation of nausea, they are not known to carry any definite pain sensation.[4] However, in a personal communication White states that he has recently found good evidence that vagi can transmit cardiac pain referred to the jaws after all the sympathetic fibers are cut. Chapman has found that pain caused by distending balloons in the duodenum and jejunum is relieved by splanchnicectomy. However, when the balloon is in the esophagus, neither total sympathectomy nor spinal anesthesia as high as the first thoracic segment stops the pain.[11] His conclusion is that this midline, diffuse, aching sensation must be transmitted over the vagi. The recent work on vagotomies in peptic ulcer has led to much speculation on the mechanism of pain relief in these cases. Alvarez, in accord with most other investigators, states that the sensory pathways are not interrupted by this operation.[9] Livingston suggests that impulses, finding themselves blocked from their customary pathways, eventually find new or previously unused pathways for reaching the sensorium.[12] Therefore, because no other pathway of pain conduction can be postulated in the case cited, and in view of the findings of White that some cardiac pain can be transmitted via the vagus, and of Chapman that some esophageal pain can be transmitted in the same way, we believe that in at least in certain instances painful impulses from the stomach may be transmitted via the vagus nerves.

Differential Diagnosis. It is beyond the scope of this communication to present the entire differential diagnosis in cases of acute abdominal crises. Certain it is that

all those acute abdominal maladies which may afflict a normal healthy young adult may also occur in a paraplegic subject. However, there are certain of these conditions to which a patient with a cord lesion is particularly liable, and it is to these entities that we should like to direct attention in this report.

In all but one of the cases presented below, comprising both complete and incomplete spinal cord transections at varying levels, abdominal pain was one of the presenting symptoms of the episode referred to as an acute abdominal crisis. In each of these selected cases, neurologic examination revealed the appreciation of touch and pain stimuli to be absent below the level of the spinal cord lesion. The site of the lesion varied in these cases from the sixth cervical to the eighth thoracic spinal segment. Nevertheless, in all but one case, abdominal pain was a presenting symptom. In practically every case the pain was described by the patient as a dull oppressive pain which was generalized or unlocalizable. In one case the pain was sharp and stabbing. In each instance, with the exception of the case referred to, the patient complained of terrible pain somewhere in the abdomen and the pain often reached excrutiating proportions but did not become localizable nor sharp. Nausea and vomiting were present in all cases, occasionally preceding onset of this unusual type of pain, and sometimes becoming so severe as to be considered the major symptom. It is our impression that paraplegic patients are more prone to become nauseated and vomit from various and sundry conditions than are non-paraplegics.

Physical signs are usually masked, inasmuch as abdominal rigidity may be absent or unrecognizable due to muscular flaccidity or spasticity, dependent upon the neurologic lesion. Abbott recently presented a report on the importance of recognizing abdominal rigidity which is often present in patients who are paraplegic due to concussion of the spinal cord.[13] He points out the fact that this rigidity of the abdominal musculature is often indistinguishable from that due to intra-abdominal pathologic processes. Tenderness was present in only one of the cases presented and is almost universally absent in these patients. Slight elevation of temperature was present in all of those cases and has been found to be of differential diagnostic value only when it suddenly spikes to 103°F. or above, in which case it has almost always been found to be due to an exacertation of the ever present urinary tract infection. The white blood count was markedly elevated in every case herein presented, and was of value only when we had previously established a "normal" base line for the patient in question. The absence of borborygmus has proved to be a particularly valuable sign when it has previously been present, thus there may be a perversion of the signs and symptoms.

When a paraplegic patient complains of pain, the neurologic lesion must first be ruled out as the producing cause. Pain resulting from a spinal cord lesion is usually radicular in distribution, and more often is sharp and knife-like in contrast to the dull, oppressive, generalized pain of which these patients complain in case of visceral disease.

The next most common cause of abdominal pain in a paraplegic, usually accompanied by nausea and vomiting, is an affection of the genitourinary tract. These patients, in practically every case, have, at some time during their course, an indwelling catheter of the urethral or suprapubic type, and in almost 100 per cent of the cases there is at least a latent genitourinary tract infection. Episodes of pyelitis are usually accompanied by severe unlocalized pain of the type already described. However, a sudden elevation of temperature to 103°F. or above usually points to the diagnosis in these cases. Inasmuch as the urine of these patients is usually loaded with white cells and bacteria, urinalysis is generally of no

diagnostic value. However, if frequent routine urinalyses are obtained, comparison with these at the time of the acute episode may reveal a change of diagnostic value. These episodes will usually respond within a few hours to chemotherapy.

An equally frequent cause of abdominal pain, accompanied by nausea and vomiting, in this group of patients is urinary tract calculus. It is well known that the atrophy of disuse leads to the mobilization of calcium with increase in the calcium content of the urine, but it must be mentioned that disuse is not alone responsible for the tissue breakdown in paraplegics. The regularity with which mild febrile episodes or operative procedures precipitate disturbances of tissue metabolism, as evidenced by loss of previously gained weight with negative nitrogen balance, breakdown of healed decubitus ulcer and rapid formation of renal calculi, suggests a disturbance of hormonal origin. While specific data are lacking in our cases, it is our impression that both anterior pituitary extract and testerone have a beneficial effect in arresting these bouts of katabolic excess.[14] Be that as it may, paraplegics are particularly prone to develop renal calculi. The pain in these cases is severe, oppressive and cannot be localized, although in the case of renal calculus presented in this report, percussion over the affected kidney caused increase in the severity of this peculiar type of pain. The temperature is usually elevated to 99° to 100°F. The diagnosis depends on roentgenologic demonstration of the calculus. In every case of suspected abdominal distress in a paraplegic patient, a flat plate of the abdomen and an emergency intravenous pyelogram are indicated. Here again, previous routine roentgenograms for comparison are of obvious value. The possibility of a perforation of the urinary bladder must be constantly borne in mind because of the friable condition of the infected bladder which develops during the course of this disease.

When the examiner is convinced that the neurologic lesion and the genitourinary tract are exonerated as the cause of the acute condition, all other causes of acute abdominal emergency must be ruled out, bearing in mind the peculiarities in the physical findings which have already been discussed. Pain may sometimes be absent in spite of an acute visceral disease. The examiner must also be aware of the patient's psychologic status. We have noted that realization of and adjustment to the paraplegic state occurs in step-like fashion; some of the steps are big ones and are frequently associated with more or less objective psychosomatic manifestations.

CASE REPORTS

CASE I. V. R., a nineteen year old white male, was rendered quadriplegic, with complete loss of motor power and all sensory modalities below the sixth cervical dermatome, on September 9, 1945. A laminectomy on March 1, 1946, revealed a complete transection of the spinal cord at the level of the fifth cervical lamina. In April, 1946, the patient began to complain of intermittent episodes of sharp stabbing pain in the mid-epigastrium. Physical examination was negative except for abdominal rigidity which had, however, been present since admission to the hospital. The pain was most marked at night and was relieved by ingestion of milk. The episodes of pain became more frequent and increasingly severe, and were occasionally accompanied by vomiting. A contrast medium gastrointestinal roentgenogram revealed the presence of a gastric ulcer. The symptoms were completely relieved by institution of an appropriate dietary regimen.

CASE II. H. K., a nineteen year old white male, received multiple gunshot wounds of the back on March 2, 1945, and was immediately rendered paraplegic, with complete loss of motor power and all sensory modalities below the eighth cervical dermatome. From the time of his hospital admission he was severely depressed mentally and had frequent episodes of nausea and vomiting, lasting about twenty-four hours. Routine studies were negative, including intravenous pyelograms, and it was believed that these episodes were psychogenic. Both the general surgical and psychiatric consultants concurred in this opinion. On March 17, 1946,

one year after injury, the patient became extremely nauseated and vomited several times. The vomiting persisted and on the second day became more severe and the patient complained of dull oppressive pain "somewhere in the abdomen." Physical examination revealed the musculature of the abdomen to be flaccid, but percussion over the right costovertebral angle increased the vague pain. The white blood count was not significantly altered from this patient's average count, and the urinalysis revealed many white cells with few red cells, but these had been present previously. An intravenous pyelogram demonstrated several calculi in the left renal pelvis and ureter with blockage of the ureter. The symptoms subsided following operative removal of these calculi.

CASE III. W. C., a twenty-five year old white male, was wounded by machine gun fire on February 24, 1945. He suffered complete loss of motor power and all sensory modalities below the seventh thoracic dermatome. He complained constantly of lancinating pain in the right upper abdominal quadrant which was not relieved by analgesic medication. On August 27, 1945, a laminectomy revealed a shrunken, non-viable spinal cord at and below the eighth thoracic segment. A bilateral radical section of the spinothalamic tracts was performed to relieve the pain of which the patient constantly complained. On November 21, 1946, this patient's abdomen became distended and the patient complained of constant dull oppressive unlocalizable abdominal pain which became increasingly severe. Peristalsis was observed to become more prominent. Enemas brought about no relief and no fecal impaction could be demonstrated. The patient did not vomit until forty-eight hours after onset. Urinalysis was unchanged and the white blood count rose to 16,000 whereas it had previously maintained itself at about 11,000. An x-ray of the abdomen revealed a marked degree of dilatation of small intestinal loops. A typical stepladder effect associated with intestinal obstruction was present. Three days after onset an exploratory laparotomy was performed and revealed many adhesions of the bowel to itself and two main adhesive obstructions, one near the root of the mesentery and one attached to the bladder. These were released and symptoms subsided postoperatively.

CASE IV. W. K., a twenty-five year old white male, became paraplegic as a result of an osteochondroma of the body of the eighth thoracic vertebra. Neurologically this patient demonstrated a complete loss of all sensory modalities below the eighth thoracic dermatome and spastic paraplegia in extreme flexion with mass reflexes. This patient complained bitterly of sharp knife-like pain throughout the lower abdomen and lower extremities. Because of the extreme flexion spasm and the constant pain, an intrathecal injection of alcohol was given, with immediate relief from both of these distressing symptoms. Any complaints of pain thereafter were adequately relieved by placeboes.

A month later the patient complained of pain in the lower abdomen which was relieved by sitting up in bed. He became extremely nauseated and vomited several times. The following day the pain was more severe and was sharp and stabbing in nature. Despite the fact that there was no somatic sensory perception below the eighth thoracic dermatome, superficial palpation over the suprapubic area resulted in exquisite pain. The diagnosis of perforation of the urinary bladder was made and confirmed at operation.

CASE V. L. L., a twenty-two year old white male, was wounded by a shell fragment on February 22, 1945, which resulted in a fracture of the eighth thoracic vertebra and complete motor and sensory loss below the eighth thoracic dermatome. He was evacuated to the United States, and by the time of his arrival at this hospital, six months after original injury, he had developed a large sacral decubitus ulcer which was subsequently closed by a plastic surgical procedure. From the time of arrival the patient complained bitterly of sharp stabbing pain in the right upper abdominal quadrant. This pain was constant but was more severe at some times than at others. The pain was not of the radicular type and it was not believed that it was due to the spinal cord lesion. An intravenous pyelogram on January 31, 1946, revealed the presence of a large staghorn calculus in the right kidney, and it was believed that this might be the cause of the patient's pain. On February 6, 1946, a pyelotomy was performed. The postoperative course was uneventful, but the pain continued unchanged in character. On March 13, 1946, a laminectomy was performed and the cord was found to be completely atrophic

below the ninth thoracic segment. The posterior roots of T-9 and T-10 were sectioned, despite the fact that the cord appeared atrophic and non-viable at this level. The subsequent course of this patient was marked by complete absence of the pain of which he had previously complained.

CASE VI. W. R., a twenty-two year old white male, was rendered quadriplegic following an automobile accident on September 9, 1946, in which the patient incurred a fracture of the body of the sixth cervical vertebra. There was a complete loss of all motor and sensory function below the level of the seventh cervical segment. On November 20, 1946, the patient became nauseated and vomited several times, being unable to retain any food or fluid taken orally. His abdomen became distended and the peristaltic sounds were exaggerated. The patient had no pain. All laboratory studies were uninformative as were x-rays of the abdomen and intravenous pyelograms. The only positive finding on physical examination was the presence of a hard fecal mass noted on rectal examination. Review of the nurses' notes revealed the fact that enemas had resulted in very little fecal evacuation during the previous ten days. All symptoms subsided following manual removal of the impacted feces.

SUMMARY AND CONCLUSIONS

1. The mechanism of visceral pain transmission is discussed and it is suggested that the vagus nerve may carry painful impulses from the stomach, and cases are presented to show that present ideas of possible pathways for pain transmission are inadequate.

2. Causes other than neurologic must be considered when a paraplegic patient complains of abdominal pain.

3. The importance and value of establishing a base line of physical findings and average laboratory data of paraplegic patients is stressed. Routine periodic checkups are essential.

4. When a definite cause of abdominal pain in these patients cannot be established, surgical exploration is justified.

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EXOPHTHALMOS*

SOME PRINCIPLES OF SURGICAL MANAGEMENT FROM THE NEUROSURGICAL ASPECT

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AS an appendage of the nervous system, the eye may present indications of highly important and significant changes in intracranial physiology. Many and varied alterations occur in the optic nerves, retinae and vessels, and the diagnosis of hemorrhages, certain neoplasms and infections may be made ophthalmoscopically. Pupillary changes and extraocular faults in innervation may permit localization and even pathologic diagnosis of disease in the central nervous system. No neurologic investigation is complete without perimetric studies. With so many and such varied evidences of neuro-ophthalmologic disturbances, it would have seemed logical if the ophthalmologists had extended their interests to the treatment of the underlying intracranial conditions and had become the neurologic surgeons of today. Evidently, the traditions of this venerable specialty and the character of training of the eye specialist were determining factors.

At any rate, the general surgeon, prompted by the neurologist, undertook the treatment of intracranial disorders. In such conditions as presented themselves, the immediate and urgent need for information on the condition of the optic discs made it necessary for the surgeon to acquire some familiarity with ophthalmoscopy and perimetry. From this beginning his interest in neuro-ophthalmology grew and of recent years a mutual interest in the treatment of exophthalmos has brought the ophthalmologist and the neurologic surgeon still closer. Ophthalmologists have made notable contributions to the neurologic aspects of perimetry. These have come largely from their intimate association with neurologists and surgeons.

In more recent years articles on the diagnosis, pathology and therapy of conditions producing protrusion of the eye have been contributed by workers from each of the specialties as well as by those from both fields working in conjunction. Many orbital lesions can best be approached from the front. This applies to those occupying the anterior third or perhaps the anterior half of the orbit and more particularly to those placed laterally. Here, a considerable space for exposure is available whereas on the nasal side of the muscle cone it is quite restricted. The principal contributions dealing with the varieties and pathogenesis of primary intra-orbital growths and the surgical methods for removal of them have come from the ophthalmologists.

Operative procedures for the surgical treatment of lesions affecting the optic pathways, chiasm, the optic nerve and the region of the sphenoidal fissure have been developed by neurologic surgeons. The intracranial approach to lesions of the parapituitary region has become commonplace in neurosurgical clinics and is similar to that needed for the orbit. Although orbital surgery includes many procedures that can be performed through the comparatively clean, but nevertheless contaminated, approach between the lids, when access to deeper structures by an intracranial route through a clean field is

required the task has fallen into the hands of the brain surgeon.

An increasing number of publications dealing with orbital surgery have appeared from both ophthalmologic and neuro-surgical clinics. The field is still a fertile one. Much remains to be accomplished and major surgery of the orbit is not widely practiced. The treatment of certain malignant or eventually potentially irremovable growths in and about the orbit is lacking in a systematic approach. The Krönlein procedure and other restricted external operations, sympathectomy and x-ray treatment, which have comprised much of the therapy, are being replaced by better planned and more thorough treatment. Orbital conditions require the attention of the ophthalmologist, and study by him in conjunction with the pathologist, radiologist and neurologic surgeon should yield valuable dividends in improved treatment.

Under the heading of orbital tumors and unilateral exophthalmos excellent articles with liberal bibliographies have come from ophthalmologists, including Benedict, Reese, Davis and Spaeth, and from neurological surgeons, notably Dandy, Davis and Martin, Love and Poppen. With such recent exceptions as those mentioned, the neurologic surgeons have concerned themselves principally with the primary intracranial lesions in which protrusion of the eye has been one manifestation. The meningiomas arising from the neighborhood of the sphenoidal ridge and fissure, both extradural and intradural, the orbito-ethmoidal osteomas of the nasal accessory sinuses and vascular lesions are among the more frequent conditions and a large literature has grown up in consequence.

The incidence of various pathologic processes is significant as a determining factor in the direction of the surgical approach to them. Benedict has estimated that intracranial involvement is present in 25 per cent of orbital tumors. Reese reports that in a large series of patients with unilateral exophthalmos the primary lesion in 63 per cent was located in the orbit.

Dandy, reporting from the standpoint of a neurologic surgeon, stated that combined orbital and intracranial tumors were present in 80 per cent of his patients. Such figures make apparent the need for the combined efforts of the ophthalmologic and neurologic surgeon in developing this comparatively untilled field.

The problem in unilateral exophthalmos is one of diagnosis and location of the lesion to determine whether it is solely intraorbital, intracranial or both. The treatment is directed toward relieving the exophthalmos by removing the cause if possible. In bilateral exophthalmos the lesion is not a removable one and decompression is required. It is frequently difficult to determine whether an exophthalmos is unilateral or bilateral. Since the normal range of exophthalmometer readings varies from 11 to 20 mm. or greater, it is not always possible to state whether or not there is abnormal protrusion and exophthalmos for that particular individual. The literature abounds in reports in which statements and judgments of unilateral exophthalmos are based on appearances instead of measurements with an exophthalmometer. Such reports are of no particular value. Because of the normal range in the position of the eyes in the orbit and the misleading appearances produced by a stare due to variation in the interpalpebral space, even experienced examiners are easily misled. Without measurement one can usually determine a difference in the prominence of the two globes by looking down on the corneas from above and comparing their positions relative to the superior orbital margin. (Fig. 1.)

The determination of whether exophthalmos is actually unilateral or only apparently unilateral but in reality bilateral is of utmost importance in diagnosis. Unilateral protrusion is due to tumor. Bilateral protrusion has a more general cause. An experienced examiner may determine by palpation whether retrobulbar resistance is normal or increased in the suspected eye and so obtain a clue as to whether the eye
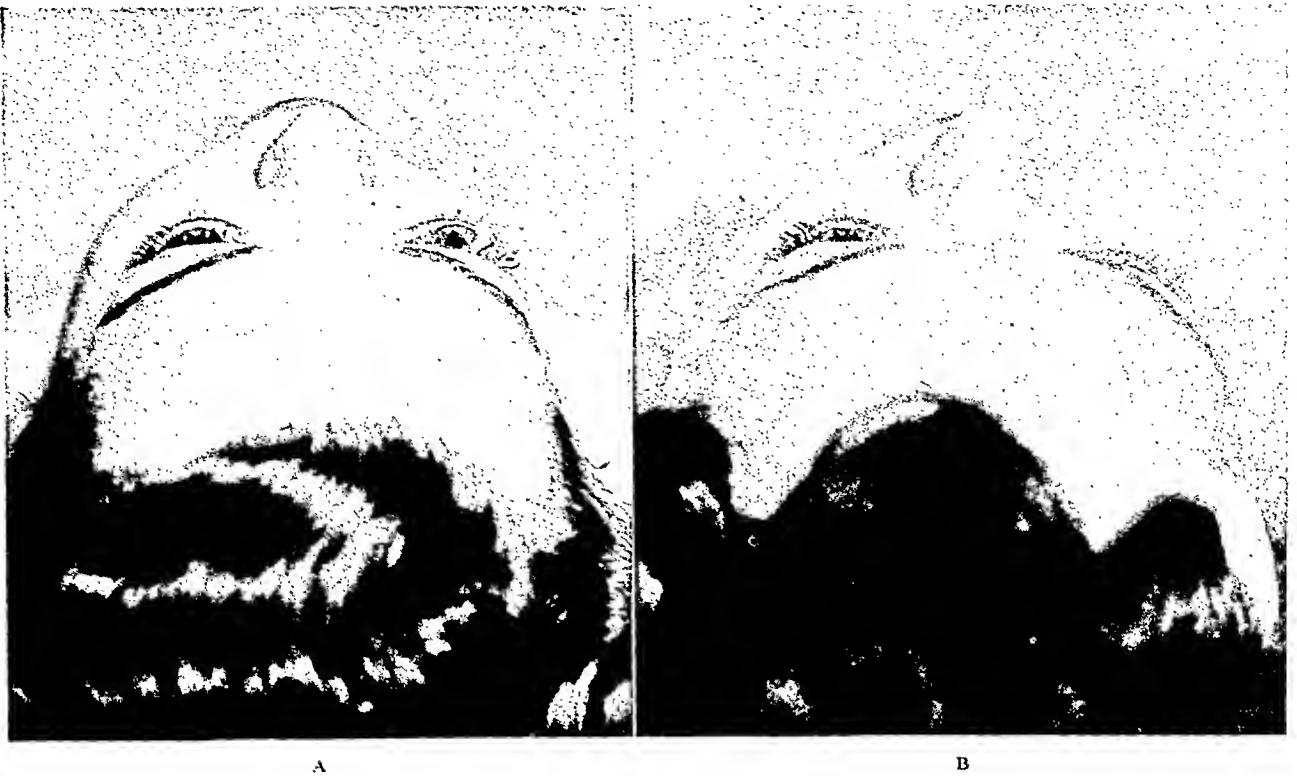
FIG. 1. A, showing relative prominence of the globes as seen from above. B, note recession of the right globe following unilateral orbital decompression.

is pushed forward or not. The response of the globe to changes in intraorbital pressure has been likened to that of a tambour.

Usually, a difference of 5 or 6 mm. between the two eyes as measured with the exophthalmometer is seen with unilateral tumors but not from other causes. In diagnosis of masses in the orbit, x-ray has come to be of increasing value, both in plain x-ray films and by angiography. Roentgenographic study should include skull and bilateral orbital views. Deformity of the orbit, alteration or loss of bone density, enlargement of the optic foramen or sphenoidal fissure and changes in the sphenoidal ridge may be seen. Commonly, the epidermoids, meningiomas and osteomas produce characteristic pictures. On this subject the writings of Pfeiffer repay careful reading. With such x-ray studies and in the absence of neurologic findings indicating an intracranial involvement, the restriction of the lesion to the intra-orbital space may be estimated. The character of the ophthalmoscopic findings and perimetric fields, the displacement of the globe, the palsies and palpation give clues

to the position of the mass in the orbit and the structures involved.

Ophthalmologic literature indicates the considerable frequency of intra-orbital inflammatory disease, often secondary to infected paranasal sinuses. Reese analyzed 174 consecutive cases of unilateral exophthalmos caused by tumors and tumor-like lesions. He excluded non-surgical exophthalmos associated with goiter, arteriovenous aneurysm, sinus mucocele, cavernous sinus thrombosis, oxycephaly and aneurysm of the ophthalmic artery. Of 109 lesions which were primary in the orbit there were: twenty-five hemangiomas, eighteen pseudotumors (chronic cellulitis or lymphogranuloma), thirteen meningiomas (these were not all primary in the orbit), ten dermoids and epidermoids, ten varieties of sarcoma, ten neurogenic tumors, nine mixed tumors of the lacrymal gland and fourteen odd types.

Unilateral lesions confined to the anterior portion of the orbit are in the province of the ophthalmologic surgeon. The operative approach is by an anterior route. Reese describes a temporal transconjunc-

tival approach suitable for laterally placed anterior growths. For those placed mesially a similar incision on the nasal side is chosen. Depending upon the site of the growth, Davis has recommended exposure through the skin at the lower margin of the orbit and Benedict one for superior tumors through the brow.

The Krönlein operation common to surgeons of sixty years ago still has followers. Its awkwardness, the visible scar which is produced, often with deformity, its inadequate decompression and the inability to extend the exposure to intracranial structures if need be leave little to recommend it and it has rightly fallen into disuse. When there is evidence that the mass is posteriorly placed or with a possibility of intracranial extension, the transcranial approaches to be described are necessary.

In 1941, Dandy published a monograph on orbital tumors. Twenty-four operative cases were reported in which he practiced a transdural operation. Surgical exposure was obtained through a frontal bone flap. The dura was opened, the brain retracted and access to the orbit was gained through the dural covering at the base and the bone of the orbital plate. This approach was necessary for most of the growths in his series. The extensive intracranial involvement required opening of the dura for removal of the growth and the invaded bone and dura at the base and into the orbit. However, Dandy used the same method and route for certain tumors which were entirely confined to the orbit. For growths confined to the posterior portion of the orbit and for decompression in bilateral exophthalmos a transcranial but extradural operation has obvious advantages.

In 1915, the writer operated upon a patient with oxycephaly who was rapidly losing vision. By a transcranial but extradural route the orbit was opened and decompressed and the optic foramen unroofed with subsequent improvement. In 1931, a similarly performed orbital decompression for bilateral exophthalmos was carried out as an extradural bilateral procedure through a coronal incision with unroofing of the optic foramen and incision of the annulus of Zinn. Surgery for bilateral exophthalmos has been required in conditions due not to neoplasm but to general systemic disturbances associated with increases in the orbital content. Since then, modifications of this operation have been made, particularly by increasing the extent of the bone removal. The wide exposure of the orbital contents and the familiarity gained by these experiences have been of help when applied to patients with unilateral tumors, for the technical methods are the same.

FACTORS IN INTRA-ORBITAL TENSION

Decompression may be required because of dangerous progression in bilateral exophthalmos and because of our inability to remove the cause of pressure. Comparison of orbital decompression with decompression for intracranial tumor does not hold for one large group of conditions, viz., progressive exophthalmos associated with thyroid disease. In these, one is not dealing with neoplastic disease nor any single gross removable lesion. The increase in orbital content is dependent upon hormonal and constitutional disturbances. Various structures, viz., muscle, fat and fibrous tissue, are all affected. Much information has been gained in recent years regarding the alterations in the amount of fat and fluids present and the myopathy, muscle infiltration and change in muscle volume in the various stages of thyroid disease. In Basedow's description of exophthalmic goiter alteration in the orbital content, notably in the size of the extra-ocular muscles, was mentioned. That alteration in orbital structures was the important and perhaps the sole factor in the proptosis of thyrotoxicosis was lost sight of over the years by reason of misdirection of attention. Claude Bernard's discovery of the rôle of the sympathetics in animals in producing pupillary changes, alteration in the width of the palpebral aperture, protrusion of the globe and vasomotor manifestations was

associated with Müller's writings on the smooth muscles of the orbit. That Bernard's experimental findings in animals could correctly be applied to man was generally accepted. Not till long afterward was it appreciated that stimulation of the sympathetics in man, contrary to the happening in most animals, does not protrude the eye. The other responses produced in animals by Bernard occur in humans, but not the proptosis.

The erroneous assumption that the sympathetics caused proptosis in man was sufficiently satisfying that during the entire period of the development of our knowledge of the causes, physiology, pathology and treatment of thyroid disease, scant attention was given to the intraorbital changes and notes of pathologic studies at autopsy were almost nonexistent. In recent years, detailed observations have been made at the operating table by biopsies and also by postmortem examinations. Moreover, laboratory research has revealed the agents capable of producing tissue changes in the orbit. These changes produced experimentally are identical with those that occur in various stages of thyroid disorders.

By use of the exophthalmometer, which measures the position of the anterior surface of the cornea with reference to a fixed bony landmark, viz., the deepest concavity of the external orbital rim, a wide normal variation in measurement is demonstrated ranging from an infrequent reading as low as perhaps 11 mm. to 20 mm. or thereabouts. A fair normal average is about 16 mm. Measurements by expert examiners vary but usually not more than 1 mm. A large series of patients with thyrotoxicosis will average about 1.5 mm. greater than normals. Contrary to earlier impressions, it has been our experience that there is further protrusion of 1.5 mm. or more in over 40 per cent of thyrotoxic patients after thyroidectomy. Recession occurred postoperatively in about 5 per cent and the remainder showed no alteration. Contrary clinical impressions are due to postoper-

ative lessening of the stare and decrease of lid retraction.

Our principal responsibility is in those instances when the exophthalmos becomes markedly progressive, particularly in the small number in which it attains a degree which endangers the patient's vision or life. The exophthalmometer readings in patients requiring operation have ranged from 22 to 35 mm. Such a measurement alone, without consideration of other factors, is no guide to the necessity for surgical treatment. In patients under observation, frequent measurements are essential to determine the progression. A full appreciation of the changes in orbital content due to alteration in solids or liquids is essential to an understanding of the surgical problem and the attendant difficulties in treatment.

From the pathologic and experimental contributions to our knowledge in recent years it is known that at different stages in the progress of proptosis there are altering proportions of orbital fat, fluids, muscle volume and lymphorrhages in the orbital tissues. Experimentally, these changes can be reproduced in animals and in fishes by injection of the thyrotropic hormone of the anterior pituitary body. All details of the complex inter-relationships of the glands of internal secretion which are involved are by no means settled.

In thyrotoxicosis, exophthalmos may be absent and other evidences of intoxication may be most severe. Again, the severity of the proptosis may be the principal concern and present the most urgent need for treatment while the nervous and circulatory disturbances may be insignificant. It is notable that severe and progressive exophthalmos may appear sometimes before other evidences of thyroid disease although progression most commonly is seen with normal or low basal metabolic rates and in postoperative patients. Progression of the protrusion by no means indicates overactivity of the gland but usually the reverse. Proptosis may appear before thyroid disease is apparent and proceed during

the stage of hyperactivity, continuing after thyroidectomy and after the establishment of a hypothyroid state. It occurs in extremes ranging from severe thyrotoxicosis to frank myxedema. It is prone to affect those in middle age and men more often than women. Most often it appears within some months after thyroidectomy.

The predominance of ocular manifestations in certain patients has been so striking and without altogether satisfactory explanation that it has afforded occasion for various recommendations in the classification of cases and for more or less complex theorizing on causation. The literature has grown rapidly under such headings as progressive exophthalmos after thyroidectomy, malignant exophthalmos, exophthalmic ophthalmoplegia (Brain), hyperophthalmopathic thyrotoxicosis (Means) and thyrotropic exophthalmos (Mulvany).

To the surgeon responsible for orbital decompression in the case where proptosis has not halted either spontaneously or with medical treatment the problem would be simple if the orbital tension were due to a solid mass. Allowing additional space would permit recession of the dangerous protrusion. Unfortunately, we are dealing with more than an increase of solids. There is a variable and fluctuating amount of fluid in the tissues dependent upon and governed by biochemical factors. The degree of retrobulbar tension and protrusion is determined by this fluid pressure and resistance of the globe to the protrusion. In the most advanced stages of malignant exophthalmos, there is an enormous increase in fluid content with tremendous extrusion of the edematous mucosa from the sclera and lids. When the tendency for fluid accumulation is so pronounced, orbital decompression allows some space for expansion and lessens retrobulbar tension but does nothing to alter the underlying biochemical factors. These persist and when allowed more space by decompression the tissues take up still more fluid.

It is for this reason that treatment by orbital decompression should not be delayed until the final stage of malignant exophthalmos is reached. On the other hand, decompression is not to be regarded as a cosmetic operation. In a few instances, bilateral progressive exophthalmos has appeared with no discoverable thyroid relationship nor other systemic disorders such as Schuller-Christian disease or hypertension. In the vast majority some connection with thyroid disturbance is clear. Usually some months after thyroidectomy these patients seek treatment although it may be many years later. As protrusion increases swelling of the lids appears. This is earlier and more marked in the upper than the lower lids. From the work of Pochin it appears that this is due to fat accumulation. Impaired motility and diplopia appear at variable stages. Photophobia, lacrimation, a watery-appearing scleral conjunctiva, scleral injection and then protrusion of the mucosa from between the lids follow. Closure of the lids is incomplete. Desiccation of the cornea, ulceration, opacities and panophthalmitis result. Before adequate treatment was available the patients in this severe state succumbed to meningitis or brain abscess.

Those patients in whom vision is threatened by corneal exposure and those in whom vision is diminishing for other reasons are suitable candidates for decompression. Vision may be threatened also by choking of the optic nerve or by pressure on the optic nerve at the optic foramen. In a few instances altitudinal hemianopsia has been attributed to this latter cause. It has been relieved by orbital decompression without unroofing of the optic foramen. Frequently, there is complaint of diminishing vision without visible alteration in the cornea or media, without choking or perimetric field changes and without a satisfactory explanation to be found in the appearance of disc or retina. Restoration of visual acuity follows decompression. Such manifestations appearing with an increasing degree of exophthalmos constitute the operative indications. Impairment of extra-

ocular movements is likely to be noted first in the upward gaze or laterally with no impairment of downward motion. In the absence of other indications, we have not considered impaired movement of the globes a sufficient reason for decompression. Often, however, it is associated with other urgent manifestations.

During the period of progression the use of thyroid substance and iodine is generally believed to be of advantage and is worthy of trial. Water excretion and lessening of water storage are promoted. X-ray therapy of the pituitary gland or of the eye muscles themselves is of unproved value although it has some advocates. Continued progression with evidence of beginning scleral edema is a danger sign and warrants immediate consideration of operative intervention.

When decompression is required, the widest possible bone removal is none too much for the desired result. Restricted procedures by the intracranial route, the Krönlein method or operation externally through brow or lids have been unsatisfactory, or if helpful have been performed in the less dangerous cases or for cosmetic indications.

TECHNICAL CONSIDERATIONS

The technical points involved in orbital decompression have become familiar to neurologic surgeons but emphasis on some features may not be amiss. These are applicable especially to those cases of endocrine origin but to a considerable extent are applicable to all extensive orbital exposures.

The preoperative and operative management for unilateral and bilateral cases are similar. Preoperative x-ray studies showing the extent of the frontal and ethmoid sinuses into the orbital plate aid one in avoiding any inadvertent opening into them.

As a first step three fine mattress sutures are used to barely approximate the lids and are lightly tied after generous lubrication of the lids with boric ophthalmic ointment. With care, no lashes are turned in. Such closure prevents subsequent protrusion of the mucosa if it becomes edematous or if edema already present increases. If corneal damage is already present, a tarsorrhaphy is in order.

When dealing with posteriorly placed tumors, with those that are both intracranial and intraorbital and for orbital decompressions, several procedures are used. For unilateral exposures we have used a scalp incision as illustrated in Fig. 13. The resulting short, midline scar is scarcely visible. A small quadrilateral bone flap is turned with the hinge in the temporal fossa. For bilateral decompression a coronal or Souttar scalp incision is made and the scalp dissected forward to the brow. The larger frontal bone flaps as used originally (Fig. 2) have been replaced by smaller ones which are ample or by triangular bone flaps having a trephine opening which is common to both in the midline as used by Poppen. (Fig. 3.) After reflection of the bone flaps the dura is peeled back from the orbital plate. For decompression the extent of the bone removal from the orbit is greater than may be required for an intra-orbital tumor. It should be wide and limited only by the extent of the frontal sinuses anteriorly and the ethmoids medially. (Fig. 4.) The posterolateral wall of the orbit is removed close up to the orbital rim after retraction of the temporal muscle. This excision is carried downward close to the antrum. Behind this the pterion and sphenoidal ridge are rongeured away. During the bone removal care should be taken to avoid opening the thin periorbita. If this is ruptured, the persistent herniation of the tense orbital content hampers the operator. It has been found unnecessary to unroof the optic foramen and incise the annulus of Zinn as was reported in our earlier patients.

Upon completion of the bone opening, hemostasis is obtained and the periorbita widely opened to the limits of the exposure. (Fig. 5.) The tumor removal or the decom-

pression with a small muscle biopsy is concluded. Upon closure the orbital content will be in contact with the frontal dura above, the dura of the temporal lobe behind and the temporal muscle laterally.

a small stab incision posterior to the operative incision and above the ear. This is removed in twenty-four or thirty-six hours.

The bone flaps are replaced and fastened in position and customary closure in layers

Line of skin incision
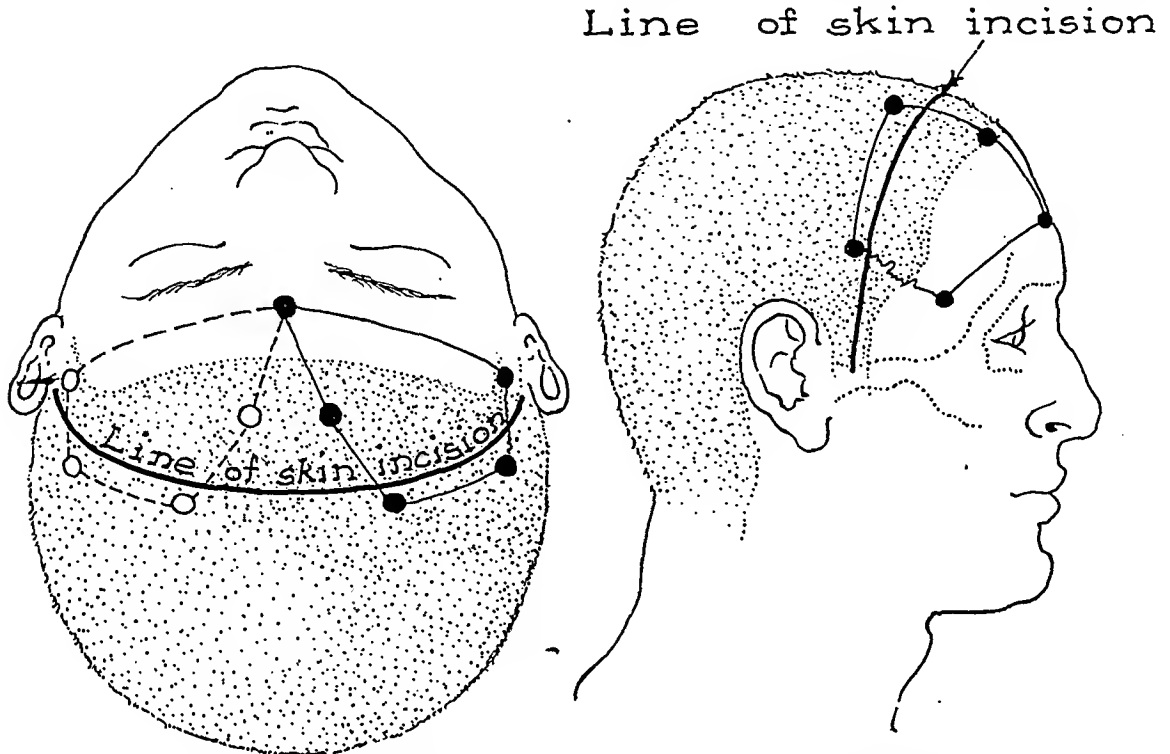
FIG. 2. Line of coronal scalp incision; outline of bone flaps originally used.

Hemostasis should be detailed. There is, moreover, less edema if a small, sewn, rubber drain is led from the pterion back beneath the temporal muscle and through

of the temporal muscle, fascia, galea and skin is made. Postoperatively, additional lubrication is applied, a cover of gutta percha tissue and a pad of suitably shaped

FIG. 3. A, triangular bone flaps introduced by Poppen for use in bilateral decompressions; B, showing unroofed optic foramen; C, large bone flaps as originally devised.

Area of bone to be removed

Area after removal of bone

FIG. 4.

Dura

covering

brain
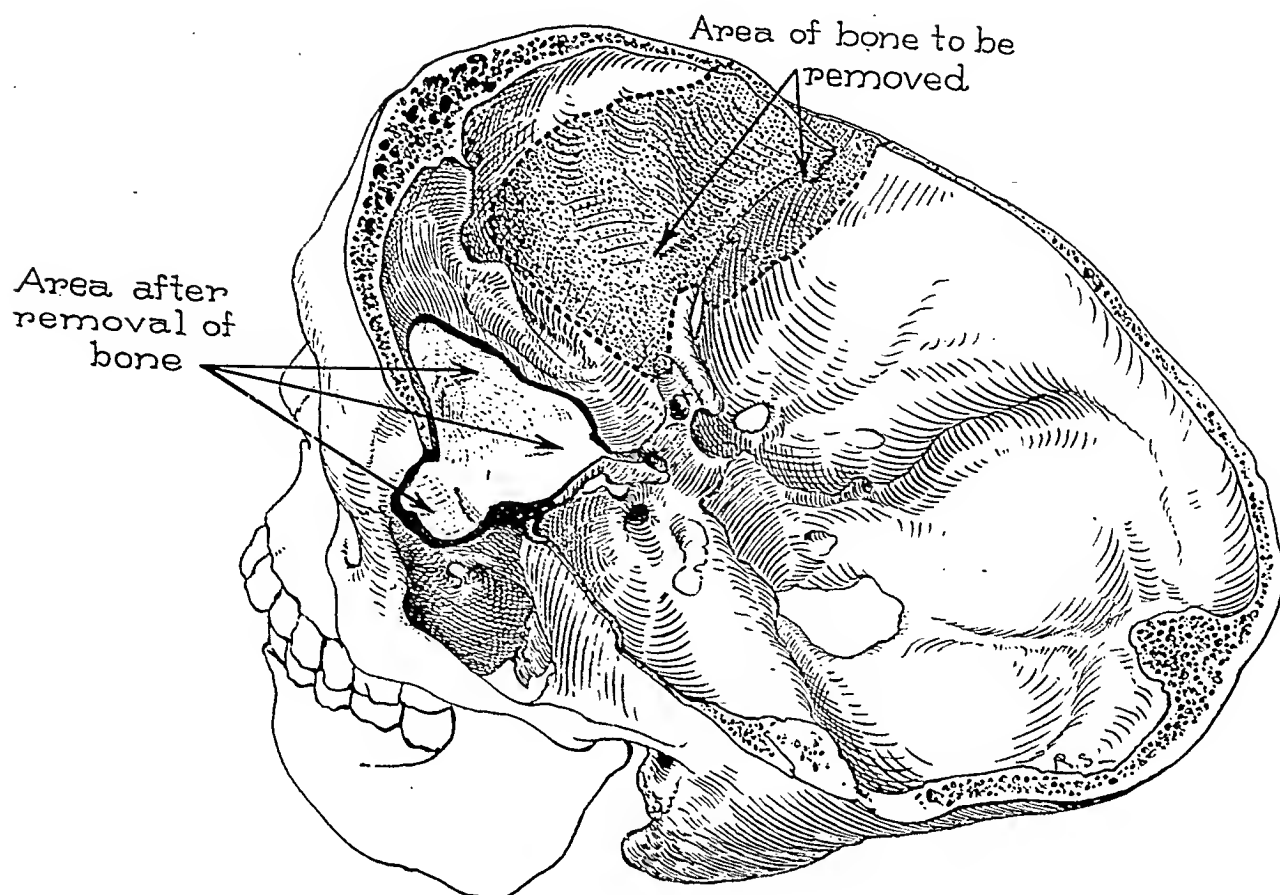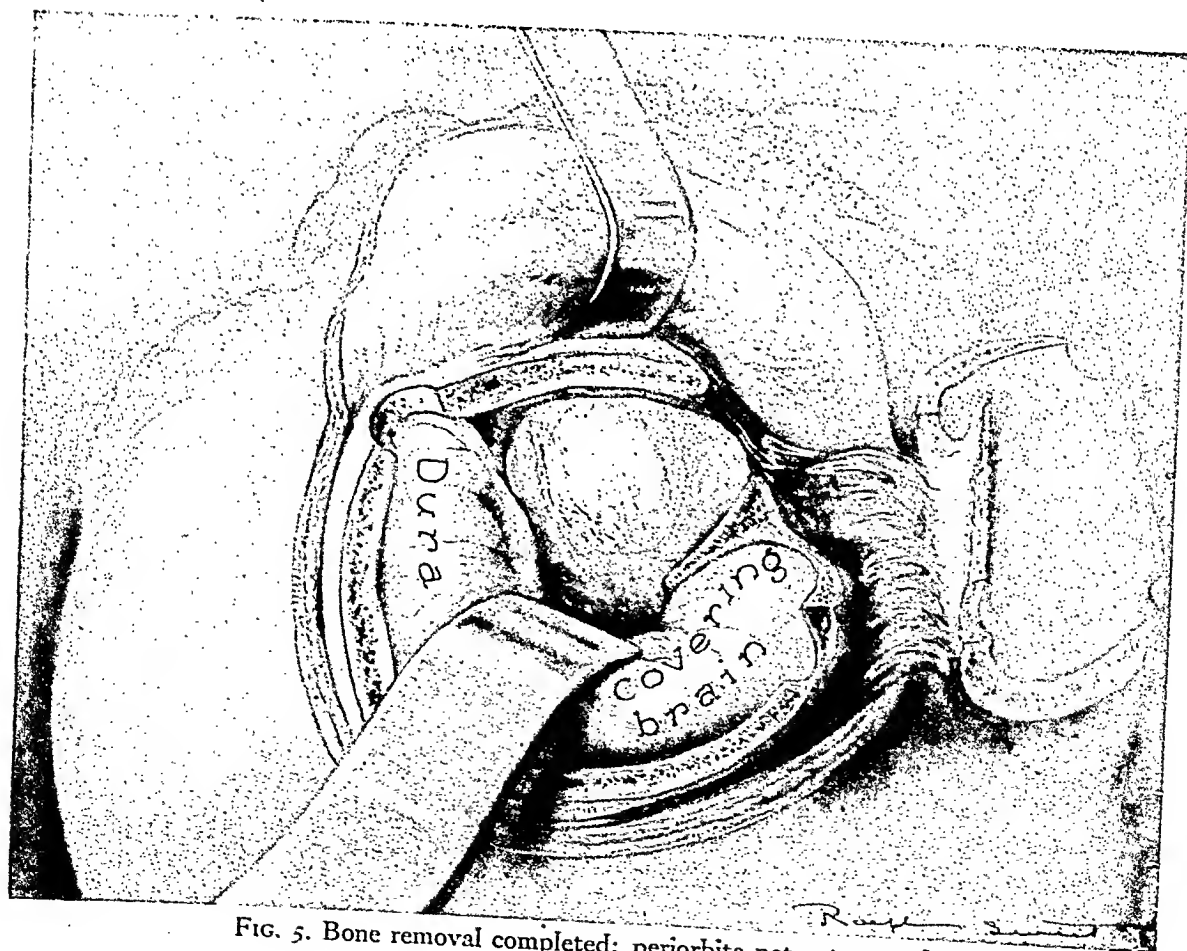
FIG. 5. Bone removal completed; periorbita not yet opened.

FIG. 6. A, B and C, advanced exophthalmos; preoperative state.

sponge rubber are placed over the lids and light compression is maintained with an Ace bandage over the head dressing. This compression is continued and the sutures are usually left in place for about a week. Before that time temporary removal of the dressing and cleansing with boric acid solution are done to remove mucoid secretion. If the mattress sutures have not been pulled and tied tightly, there should be no infection about them.

The postoperative treatment, barring such complications as may be common to all frontal operations, is simple or prolonged depending upon the protrusion and edema of the scleral and palpebral mucosa. Ordinarily, and with the precautions mentioned, there will be little trouble if there has not been some preoperative extrusion. If edema is present and pronounced, it is apt to be continued for some time and require a prolonged hospital stay. Care of edema must be closely supervised by medical personnel. Removal of secretion with 2 per cent boric acid solution and lubrication with boric ophthalmic oint-

FIG. 7. Postoperative stages of patient shown in Figure 6. A, recession of globes at conclusion of operation; B, protrusion of edematous mucosa one week later.

FIG. 7. C and D, condition at end of four weeks; E and F, one year after operation.

FIG. 8. Edema in unoperated patient.

FIG. 9. A and B, preoperative state; protrusion and scleral irritation and beginning edema; C and D, postoperative result.

ment are carried out with avoidance of all irritating washes or drops. A goggle or perforated shield prevents drying. Resuturing of lids after replacement of the mucosa may be needed. Limitation of fluids and salt, use of thyroid substance and spinal puncture are tried but the results of these measures are not impressive.

Immediately following operative decom-

pression, the ocular recession is marked. Pulsation of the globe transmitted from the dura is readily evident. Shortly, due to edema, there will be return of some protrusion. The pulsation will diminish but will persist in some measure over a period of months. The patients are rarely aware of it and have no visual disturbance as a result.

Swollen optic discs and retinal hemorrhages are present in the minority of patients presenting themselves for operation. In our first experiences, it was thought advisable to unroof the optic foramen for relief of the choking. Later experiences show this is unnecessary. The swelling disappears rapidly after decompression.

Permanent recession of proptosis of from one or two to several millimeters follows decompression. Progression has not occurred postoperatively in our patients. Impaired ocular movements improve markedly but some fault in movement and diplopia may persist.

Bilateral orbital decompression may be performed at one operation and this has been done in most of our subjects. It involves a longer operative procedure and might be criticized on the greater risks of a complicating infection. When great edema

A   B

FIG. 10. A, preoperative condition; B, postoperative result.

Fig. 11. Preoperative appearance and postoperative result three weeks later.

of the mucous membrane is present with or without corneal ulceration and considering the period with eyes bandaged, a unilateral operation is sometimes decided upon.

In the postoperative period, edema of the scleral and palpebral mucosa with protrusion from between the lids has been the most frequent concern. When present before operation, edema is apt to be exaggerated for a postoperative period. This applies to patients with progressive bilateral exophthalmos. (Figs. 6 and 7.)

Few other complications have been met with in thirty-nine cases of this type. Rhinorrhea occurred in three patients due to opening of the ethmoids. In one instance, a pneumococcic meningitis followed but was controlled promptly by chemotherapy. In all patients the rhinorrhea disappeared

in a few days. Marked retraction or elevation of the dura may be followed by frontal lobe signs such as disorientation and confusion over a short time. In this series there have been no deaths. In a few patients operation on one side only was required.

Operation for unilateral intra-orbital tumor differs in no essential way from that described above for bilateral decompression. A small quadrilateral frontal bone flap is preferable to a triangular one and orbital bone removal need not be as extensive. Suturing of the lids prior to operation and a light compression bandage following it are helpful.

Since the transcranial extradural operation was devised, other methods of decompression have been suggested and reported in small series of cases. Kistner has decom-

A   B
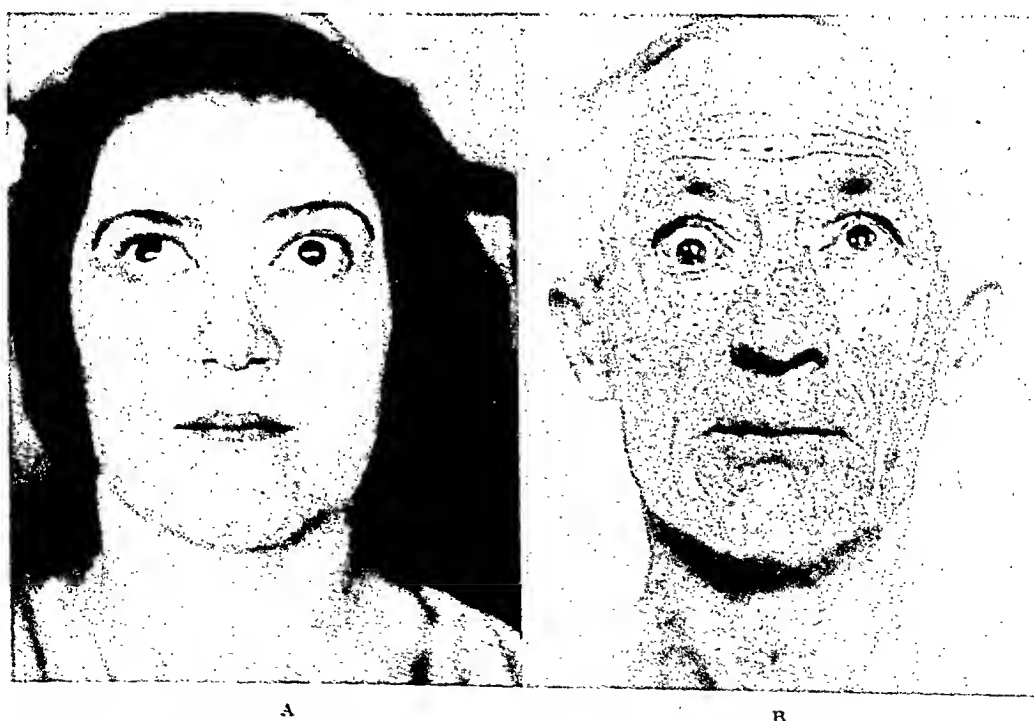
Fig. 12. A and B, examples of so-called "exophthalmic ophthalmoplegia"; impaired motility, moderate protrusion; no evidence of thyroid disturbance.
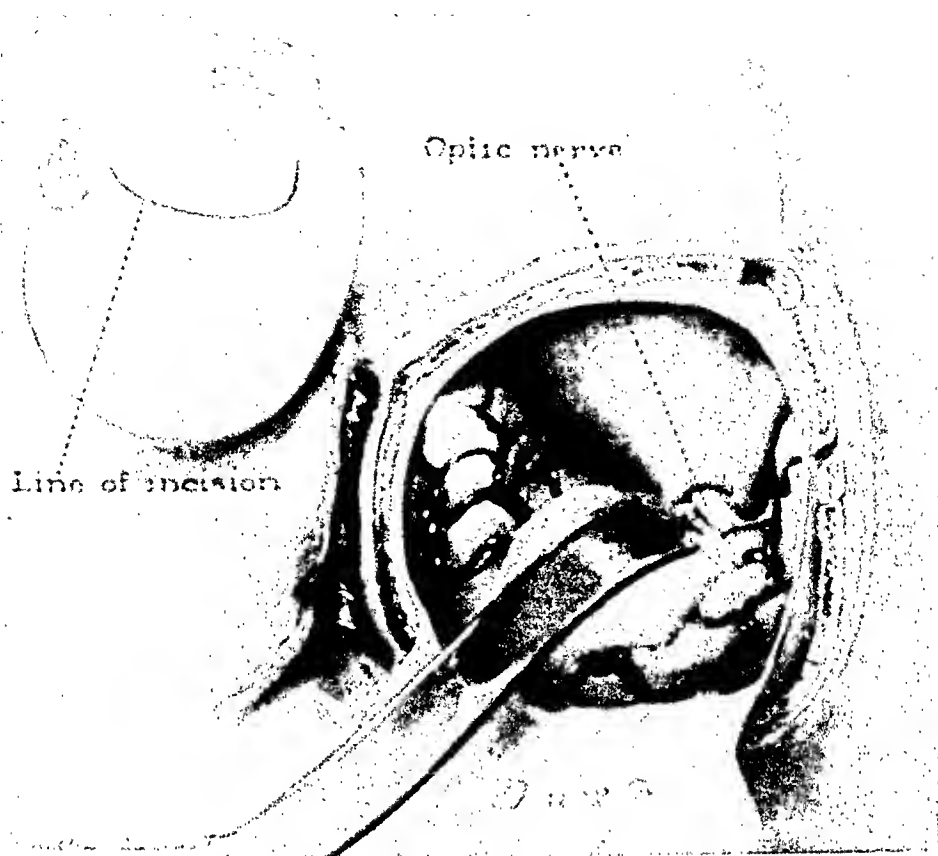
Fig. 13. Frontal pole retracted exposing orbital plate, sphenoidal ridge, optic nerve and chiasm.

pressed the orbit into the frontal sinuses with good results. Bothman, Sewall, Swift and others have practiced or advocated removing the bony wall of ethmoids or antrum for the same purpose. The writer has had no experience with these. In our patients the largest decompression possible has been sought through a clean field. The risk has been minimal and the procedure should leave no visible scar or deformity.

Guyton, in a discussion of decompression of the orbit, advocates a skin incision through the hair line and a comparatively small lateral or posterolateral decompression of the orbit. Such an opening in our experience, although restricted as a decompressive operation, serves a valuable purpose. He recommends it as suitable for cosmetic purposes as well as for the more dangerous proptosis with which we have dealt.

For combined intra-orbital and intracranial lesions one of two courses may be followed. A larger bone flap than the small triangular one is required. If the orbit is explored first, the intracranial lesion may be pursued by opening the dura along the sphenoidal ridge. If an intradural growth is known preoperatively to be present, the dura is opened over the frontal pole which is then retracted (Fig. 13) for a radical procedure and the concern is not primarily with the exophthalmic manifestation.

For exposure of the posterior and lateral portion of the orbit and visualization of the optic nerve and its neighborhood intracranially a temporal approach has a useful place. A scalp incision as shown in Figure 14 is made as well as a similar one in the temporal fascia and muscle. A small fringe remains attached to the upper margin of the temporal fossa for later resuturing. With a periosteal separator, the muscle is cleaved off from the temporal fossa and the bone removed. (Fig. 14 and 15.) By opening the dura and sucking out fluid
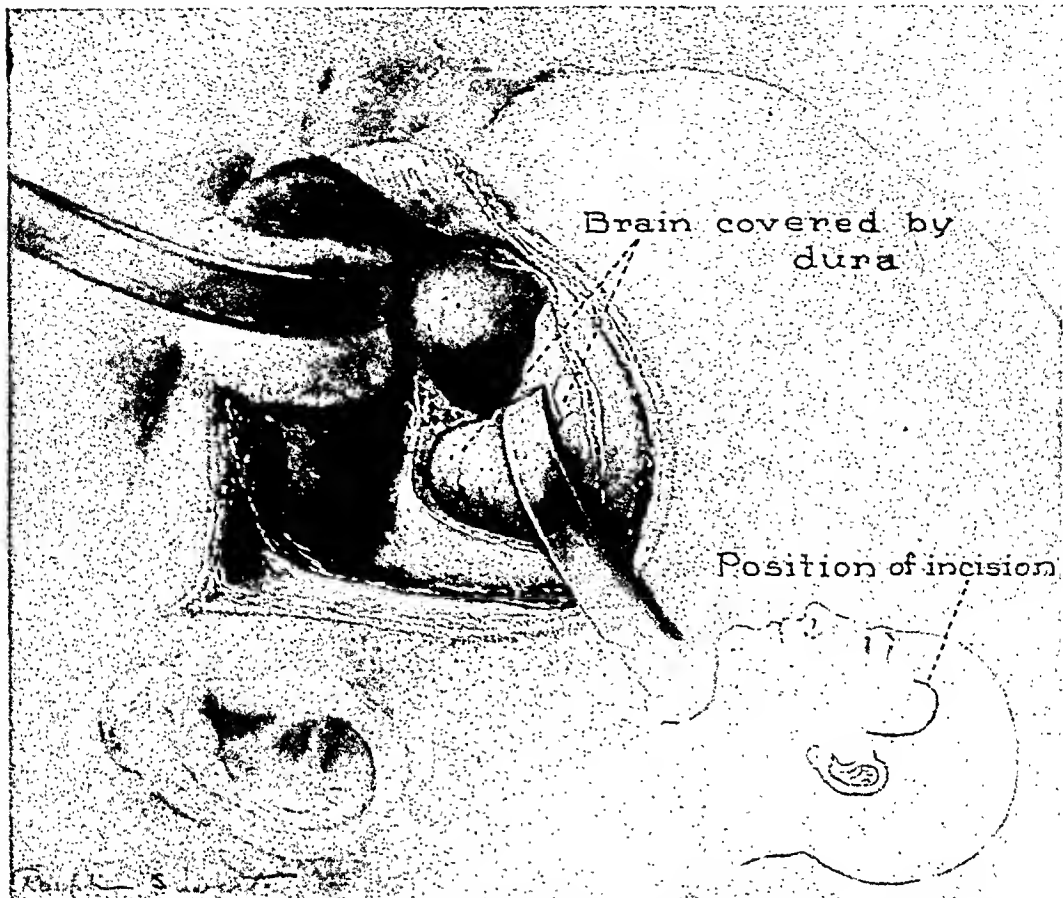
FIG. 14. Line of incision (insert); temporal musele retracted; bone removal with periorbita and dura of the frontal and temporal lobes exposed.
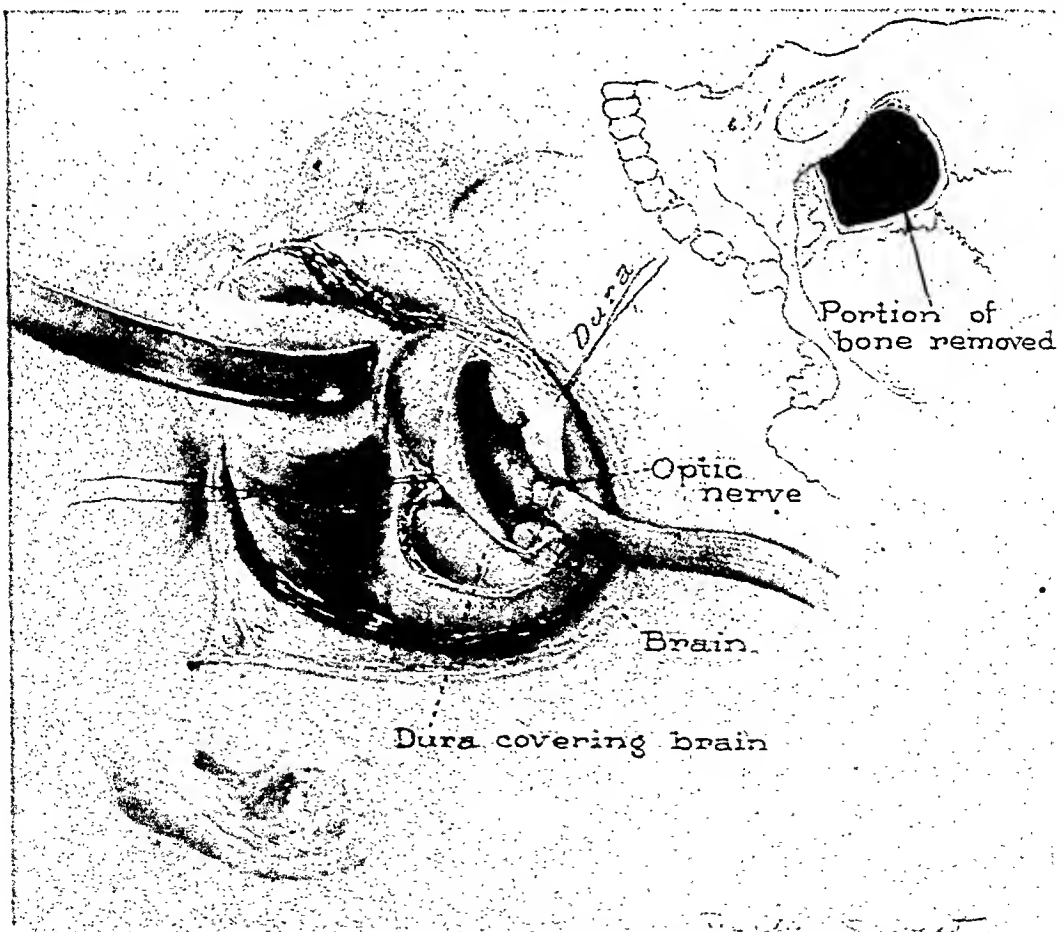
FIG. 15. Intracranial exposure through temporal approach.

intracranial exploration is readily made. This has been found useful in determining involvement of the optic nerve prior to intra-orbital enucleation. It has been highly satisfactory for the laterally placed tumors such as cholesteatomas.

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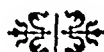
COMPLICATIONS ACCOMPANYING SURGICAL RELIEF OF PAIN IN TRIGEMINAL NEURALGIA

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THE diagnosis of major trigeminal neuralgia in the typical patient is easy. An elderly patient presents himself with sudden, intense, lancinating pain in one or the other side of the face. This pain usually affects the upper or lower lip and extends up to, but never across, the midline. The pain is brought on by any local irritation such as talking, eating or washing the face. The pain is definitely paroxysmal in character, coming on in intense, stabbing waves with little or no abnormal sensation between attacks in the area involved.

As a rule, the patient has had a short, initial spasm months or years before the surgeon sees him and, between subsequent attacks, has noted periods of spontaneous remission during which there has been no abnormal sensation of any kind in the face. The presence of a trigger zone is characteristic. For example, if the upper and lower lips are involved in the pain, it is frequently noticed that touching either area will cause a spasm of pain to pass from the lower half of the face into the upper half and the patient soon learns to avoid touching this trigger area. Not infrequently, the experienced observer makes the diagnosis of major trigeminal neuralgia as the patient steps into the office. He will notice that part of the face about the upper lip and the ala of the nose is unwashed, dirty and greasy. This is the trigger zone. Any attempt on the part of the patient to clean up this area results, at once, in the lancinating, stabbing, paroxysmal pain so characteristic of this disease.

Fortunately, both sides of the face are but rarely involved. In a series of, roughly, sixteen hundred cases of verified major trigeminal neuralgia, there have been but two in which the pain was noted to be bilateral at the same time. There have been forty-three cases in which, after the pain had occurred on one side, this attack was followed a number of years later by similar pain on the opposite side.

The treatment of major trigeminal neuralgia has become completely standardized. Section of the sensory root of the trigeminal nerve is the accepted method for producing permanent relief. Unquestionably the relief from the intolerable, stabbing, lancinating, paroxysmal pain characteristic of this condition is complete. Nevertheless, the patient is penalized for relief from this pain. Any wide experience with the results of the treatment of major trigeminal neuralgia by root section will soon convince the honest and competent observer that while, for the most part, section of the sensory root gives the patient the freedom from pain that he expects, nevertheless, he pays a certain price for complete relief of pain which, in some instances, is rather higher than had been anticipated.

The price that the patient pays for the complete relief of pain is total anesthesia in the areas to which the pain was originally referred. This anesthesia is produced by section of the sensory root, whether the root be cut at the pons by the occipital approach or just behind the ganglion by the temporal route. Dandy[1] claimed as one of the advantages of the occipital approach to the sensory root that, by this technic, he was able to cut just the lateral fibers of the root and thereby bring about a selective relief of pain alone, without loss of touch sensation. Our experience has been entirely at variance with Dandy's. No matter where the sensory root is cut, com-

plete loss of all modalities of sensation was produced if pain sensation was abolished. Occasionally, when a subtotal section of the root was performed, total anesthesia in the third division for all modalities of sensation and a partial loss of sensation in the second division resulted. However, in the second division, where sensation was partially preserved, it was always impossible to determine that pain sensation was lost to a greater degree than touch. It is quite true that the operation devised by Sjöqvist,[2] the section of the descending root of the trigeminal nerve in the medulla, has produced selective loss of pain sensation without an equivalent loss in touch sensation. We were enthusiastic about the possibilities of the Sjöqvist technic until experience showed that, unfortunately, the fibers of the third division, which is very commonly involved in most severe cases of major trigeminal neuralgia, lay most centrally in the medulla. A deep, bold section in the medullary area was necessary to assure section of these centrally situated third division fibers. The consequences of a deep section in this area was sometimes quite devastating for the patient. A very marked ipsilateral ataxia and dyssynergia of a permanent nature can be produced by too deep a section of the medulla in attempting to carry out the Sjöqvist technic. Medullary tractotomy was carried out in six patients with typical major trigeminal neuralgia. In five of these six patients, the pain recurred and root section was necessary to relieve the pain permanently. The sixth patient has been entirely comfortable for the past seven years. Very little, if any, loss of touch sensation is demonstrable in this patient although pain has been entirely lost and his major trigeminal neuralgia completely relieved.

One important consideration exists when the surgical treatment of major trigeminal neuralgia is suggested. It must always be remembered that facial neuralgia is not a lethal disease. It may make the patient extraordinarily uncomfortable and unhappy but as far as our records show, there have been only two cases of suicide because of this dreadful facial pain in our series of some sixteen hundred patients. Consequently, when surgery is considered the procedure used for relief of pain must be that which in the surgeon's hands carries the lowest mortality. As has been stated, there are two routes of approach to the trigeminal afferent fibers; one, through the temporal fossa, either intradural or extradural, with section of the root immediately posterior to the gasserian ganglion; and secondly, through the posterior fossa, either cutting the sensory root where it leaves the pons or severing the descending fibers of the trigeminal nucleus in the medulla. Either of these surgical approaches can be carried out with the patient in the sitting position. It is certain that the transtemporal approach lends itself a little more readily to the use of a local anesthesia than does the occipital approach. Dandy,[1] who originated the occipital approach, carried his procedure through with the patient in the prone position. When it is remembered that 85 per cent of the patients who are operated on for major trigeminal neuralgia are over sixty years of age, with all the possible complications accompanying advanced years as hypertension, arteriosclerosis, myocardial or renal disease, the disadvantages of the prone position, with the need for endotracheal or rectal anesthesia, at once become obvious. The exposure of the sensory root by the occipital approach is rather more difficult than by the temporal approach. The root lies more deeply. If bleeding occurs, consequent upon rupture of the petrosal vein which runs from the cerebellum to the lateral sinus directly in the line of this approach or if, as is not uncommonly the case, an artery accompanies the sensory root and is injured at the time the root is sectioned, the control of hemorrhage in this deep field is a matter of considerable difficulty. Electrosurgery and the use of fibrin foam and other coagulants have rendered the hazards accompanying hemorrhage much less great. The close proximity of the brain stem and

the facial nerve makes the careful and conscientious operator very nervous if he finds himself involved in bleeding in this dangerous area. A medullary tractotomy certainly cannot be carried out under local anesthesia. When the incision is made into the medulla to section the descending root, the patient suffers intense, lancinating pain and is quite likely to jump and twist. The depth to which the incision into the brain stem should penetrate is a matter of millimeters. Any sudden movement on the part of the patient can very well involve him in serious consequences in spite of the best efforts of the operator. It is our very distinct impression that the erect position is the safest for section of the sensory root, whether this be carried out through the temporal or through the occipital approach. Certainly the consecutive series of cases operated on by Frazier[3] and Cushing[4] without a mortality has never been surpassed. However, their success was not due necessarily to position because Cushing operated on his patients in the lateral, horizontal position whereas Frazier always employed the sitting position. Both of these operators used very light, open drop ether, given by nurse anesthetists who were skilled and experienced in carrying these patients through this operative procedure with a minimum of anesthesia. The modern, young, medical anesthetist knows how to give a dozen different types of anesthesia but, in our experience at least, he is deficient in his knowledge of the value of small amounts of open drop ether. In sensory root section no relaxation is necessary. All the operator asks of the anesthetist is to keep the patient sufficiently under the anesthetic so that he does not move. As soon as the root is cut the anesthesia can, of course, be stopped. We have seen skilled nurse anesthetists carry elderly patients along for two hours on less than 3 ounces of ether and have them so lightly anesthetized throughout the operative procedure that they would respond during the closure of the wound. It is difficult to persuade the modern, medical anesthetist to give open drop ether in this fashion.

Recent experience has shown that elderly patients who have been given morphine preoperatively are likely to suffer a serious fall in blood pressure when placed in the erect position. There has been at least one recent fatality in our clinic which we attributed to the preoperative administration of $\frac{1}{4}$ gr. of morphine. Consequently, we have recently eliminated morphine as a preoperative medication, substituting a small dose of phenobarbital plus atropine. Our overall mortality in major trigeminal neuralgia, using the erect position and the "transtemporal" extradural approach, is 1.85 per cent. We would repeat that since major trigeminal neuralgia is not a lethal disease, if an operative death occurs in an attempt to relieve it, the surgeon has unquestionably predeceased the patient unnecessarily. It is for this reason that we insist that every precaution should be taken to protect these elderly patients who have major trigeminal neuralgia from any operative accident, whether that accident be due to the surgical technic used or to the anesthesia employed.

A second complication, which can mar the results of section of the posterior root for relief of the stabbing, lancinating pain produced by major trigeminal neuralgia, is a paresthesia of the face in the anesthetic area. In our series, 62 per cent of the patients were completely and entirely satisfied with the operative results. Thirty per cent were very glad that they had had the operation and had been relieved of their pain but stated that they were conscious of the numbness of the face. They accepted this numbness quite philosophically as the price they had to pay for relief of pain. The remaining 8 per cent complained of the numbness in their face; about one-half of these, or 5 per cent of the total, complained with very considerable bitterness. The face burns, it has a creepy, crawling sensation in the numb area; the tongue and lip feel as though they had been dusted with pepper and are hot and raw.

These patients insist that the operation, while not a complete failure, has not by any means lived up to their expectations as far as relief from pain is concerned. An occasional patient will state that he is more uncomfortable after operation than he was before because this burning sensation in the face is present for twenty-four hours a day and keeps him awake at night, whereas the lancinating pain was only intermittent, rarely occurred at night, and between attacks, which he could avoid by precautions to which he had become accustomed, there was no change in facial sensation. The possibility of paresthesia in the anesthetic area is the principal objection to sensory root section. It was for this reason that we hailed with so much hopefulness the reports of medullary tractotomy described by Sjöqvist, with its production of selective analgesia without complete anesthesia. However, we have become completely dissatisfied with the Sjöqvist procedure because, in our experience with six patients with major trigeminal neuralgia, two complained of paresthesia and five had eventual recurrence of the trigeminal pain.

It is our firm opinion that the best method for preventing paresthesia in the face following root section is by preliminary relief of the neuralgic pain by alcohol injection of the appropriate branch, or branches, of the nerve involved. We appreciate fully that preoperative block of the second or third division with alcohol is a painful ordeal for the patient. The nerve cannot in every instance be accurately injected and the relief produced is not permanent. However, following a successful block, the patient can be relieved of pain for a year to eighteen months and a second block will keep him pain-free for another year. More important, the anesthesia resulting from the alcohol block is precisely similar to that which will be produced by section of the sensory root. If, therefore, a patient has had his pain relieved by alcohol block, he knows how his face will feel and appreciates very fully the price that he must pay for permanent relief of pain. After the anesthesia, consequent upon alcohol block, wears away, the pain recurs. When the patient returns asking for complete relief, he may then be told that he can be permanently relieved by root section but that he will always have the anesthesia. If, during temporary relief by block, the anesthesia has not been an annoyance to him, he is quite willing to have the sensory root cut and the anesthesia made permanent. If, after an alcohol injection, he objects to the numbness of his face, he can be told that if he will be patient for a year that numbness will disappear, sensation will return to normal and his pain will recur. However, once the sensory root has been cut, the anesthesia produced is permanent and if the patient objects to it nothing can be done to improve the situation. It is for this reason that we believe that preparatory alcohol injection, in spite of its disadvantages, has a very definite place in the treatment of major trigeminal neuralgia. Our experience has been that those patients who have had preliminary block of one or the other branches of the nerve are much less likely to complain of the permanent, total anesthesia which follows section of the sensory root of the nerve and consequently are less likely to develop burning paresthesia and other disagreeable sensations in the anesthetic area.

As far as the first and second divisions are concerned, other methods for temporary relief of pain are available than simple alcohol injection. Avulsion of the supraorbital nerve through an incision in the eyebrow is a simple, safe procedure and will relieve the supraorbital neuralgia for four or five years. An incision through the mucous membrane under the upper lip will expose the second division very readily. Once the infraorbital foramen with the emerging nerve is visible, it is a simple matter to introduce a wire for 2 or 3 cm. into the infraorbital canal and coagulate the nerve with the electrosurgical unit. In a nervous patient who insists upon an anesthetic for relief of second division neuralgia, this procedure has some ad-

vantage over direct alcohol injection. As far as the third division is concerned, if the lower lip and teeth alone are involved, the nerve can be blocked with a drop or two of alcohol where it descends around the ramus of the mandible. If the tongue is involved, however, the lingual branch of the third division usually leaves the main trunk above this point. Consequently, alcohol injection through the cheek, picking up the whole nerve where it leaves the skull is necessary when the paroxysms of pain involve the tongue.

The third complication of section of the sensory root, particularly when it is completely sectioned, is keratitis and ulceration of the cornea in the ipsolateral eye. Subtotal avulsion of the sensory root, as suggested by Frazier[5] in 1925, sparing that part of the root that runs to the first division and thus not completely desensitizing the cornea, has well nigh eliminated this particular complication. The problem always arises, of course, in those patients who have major trigeminal neuralgia in all three divisions, as to whether a subtotal avulsion is appropriate. It is our definite experience that it should be done unless the supraorbital area is the trigger zone. Fortunately, this is the exception rather than the rule. In those instances when the trigger zone lies in the second and third division and when irritation of this trigger zone results in pain in the first division, it is entirely safe to carry through a subtotal avulsion. Desensitization of the trigger zone will eliminate the spread of pain into the first division. However, in certain cases when a subtotal avulsion has been done, there is, later on, a spread into the supraorbital area. If this occurs, it is a simple matter to avulse the supraorbital nerve at its foramen. It is, in our opinion, always much better to preserve the fibers in the root running to the first division than to do a total section. As we have said, if pain spreads later on into the first division, it can be relieved for four or five years by avulsion of the supraorbital nerve. Many of these patients are elderly and much can

happen in five years. In our series there have been between 5 and 10 per cent of recurrences in the first division, usually after six or seven years have passed. It is much better, in our opinion, to accept the risk of recurrence in the first division consequent upon sparing the inner fibers of the sensory root than it is to avulse completely the sensory root and risk the possibility of complications in the cornea.

Prior to 1925, when total avulsion of the sensory root was the routine procedure, the incidence of some degree of keratitis was 15 per cent. In about one-half of these patients, closure of the lid was necessary to control the keratitis and in between 4 and 5 per cent of the total patients the eye on that side was lost. This calamity usually followed neglect of the eye on the part of the patient and attending physician. The keratitis spread, burrowed in to involve the anterior chamber, produced an anterior hypopyon and enucleation of the eye was necessary. At the present time most competent ophthalmologists recognize this situation promptly and close the eyelid. Closure of the eyelid will result in healing of the keratitis within ninety-six hours. Tarsorrhaphy is the only proper treatment for this complication.

The final complication which may accompany operation on the sensory root by either the temporal or occipital approach is facial paralysis. In the last five hundred patients operated on through the temporal approach, this complication has appeared in, roughly, 6 per cent of patients. Fortunately, in none of these patients has the paralysis been of a permanent nature. In every instance it has disappeared within six months. When the sensory root has been completely avulsed, the appearance of a facial weakness creates a very awkward situation. Inability to close the ipsolateral eye following complete section of the root, with total corneal anesthesia, will certainly result in drying of the cornea and consequent ulceration. Closure of the lid is therefore necessary in all these patients. The lid should be kept closed until

there has been sufficient return of power in the facial nerve to assure closure of the eyelid. If subtotal avulsion has been done and if corneal sensitivity is retained, tarsorrhaphy is not necessary. However, very close watch should be kept on the eye and if there is any slightest evidence of keratitis, lid closure is imperative.

The cause for facial paralysis or weakness following approach to the sensory root by the temporal extradural route is injury to the superficial petrosal vein or the vidian nerve. This nerve and vein run from the facial canal along the anterior face of the petrous ridge and under the ganglion. For this reason during the separation of the dura from the ganglion sheath, it is necessary to take care that the dura is not stripped from the anterior surface of the petrous ridge. The dissection should be made in such a fashion that the root can be exposed without elevating the dura in this area. If the dura covering this nerve and vein is not disturbed, facial paralysis is much less likely to result. The avoidance of facial paralysis during an approach to the sensory root through the temporal fossa is one of the claims made for the intradural approach. Facial paralysis or weakness can also follow the occipital approach to the sensory root. However, during this approach it is rather more likely that the facial nerve is seriously injured with a greater probability of permanent and lasting facial weakness.

In summary, it can be said that while section of the sensory root will relieve permanently and completely the savage, paroxysmal pain of major trigeminal neuralgia, nevertheless, both surgeon and patient must realize that for relief of pain the patient pays a definite price. The basic, inescapable price that he pays is complete anesthesia of the face. The surgeon's skill in selecting patients for operative procedure and his deftness in carrying out the surgical technic can eliminate all of the other complications herein enumerated. It is important, however, for both surgeon and patient to realize that, while the operative procedure for the relief of major trigeminal neuralgia can and will relieve the paroxysmal pain, nevertheless, the pathway to complete satisfaction with the operative procedure on the part of the patient is strewn with certain definite hazards.

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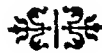
HYPERTENSION AND ITS SURGICAL TREATMENT BY BILATERAL SUPRADIAPHRAGMATIC SPLANCHNICECTOMY*

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HYPERTENSION presents the most serious medical problem before us today. No other disease exacts such an appalling annual toll. Its final manifestations of either cerebral, cardiac or renal damage account for more than 200,000 deaths each year in our country alone. Few realize that nearly all cases of so-called cerebral accidents, which include both intracranial hemorrhages and cerebrovascular thromboses, most cases of heart disease, including those with coronary thrombosis, and the great majority of cases of so-called Bright's disease have as their primary factor, hypertension.

The financial loss due to necessarily curtailed physical and mental activities resulting simply from the distressing symptoms of hypertension, and the actual loss of productive time due to the semi-invalidism of many victims of hypertension reach staggering figures. When evaluating the enormity of this disease, we must not forget the amount of suffering it causes, headaches, nervousness, irritability, insomnia, nocturia, shortness of breath and the all too frequent clouding of mentality. Any treatment which, with reasonable safety, can reduce this enormous death toll or can alleviate in a worth while degree the distressing symptoms and prolong the life of these patients is certainly worth while. Medical treatment has signally failed in many cases and we believe that surgery today offers far more than any other type of therapy a real hope of attaining these ends.

Considerable confusion has arisen because of the various designations of hypertension. We prefer the term arterial hypertension but commonly the terms essential or benign hypertension are used. Both are misnomers because it is not essential, as was at one time believed, to have an increased blood pressure in order to force blood through a possibly inadequate arterial system; and the disease is never benign although serious manifestations may not appear for many years.

The blood pressure may be temporarily or continuously elevated in a wide variety of conditions, for example, in cerebral trauma the blood pressure may rise very rapidly with the increase in intracranial pressure either from hemorrhage or edema. Certain tumors as basophilic adenomas of the pituitary gland and some adrenal tumors can produce either a continuous or recurrent elevation of blood pressure and rather late manifestations of chronic nephritis may also produce hypertension. Coarctation of the aorta, while uncommon, should always be considered a possibility in any patient, especially in a young individual who is found to have hypertension. This is a very brief list of the possible causes of elevated blood pressure. Such a list comprising forty-seven pathologic conditions associated with an elevated blood pressure was compiled by Page[11] in 1939. The diagnosis of arterial or, as it is more commonly called, essential hypertension, is therefore made by the exclusion of the other conditions which may present a similar picture.

Arterial hypertension is a highly complex disease syndrome characterized at first by only a mild elevation in the systolic

and diastolic blood pressures. From then on its manifestations are many and the progress of the disease varies greatly. One patient may have more or less incapacitating symptoms of headache, nervousness, insomnia and ease of fatigue but without evidence of cardiac or renal damage. Another with apparently the same elevation in blood pressure may be practically symptom-free but will shortly begin to show evidences of cardiac disease. Still another patient may have the usual hypertensive symptoms and some evidences of cardiac involvement but on careful examination it is found that the kidneys are chiefly affected. Another patient may first manifest evidences of this disease by a so-called stroke, either a cerebral hemorrhage or a cerebral arterial thrombosis.

The rapidity of progress of the disease varies as much as its manifestations. A few live out a normal life span without any untoward incident but the majority sooner or later have serious constitutional changes with cardiac, renal or cerebral involvement. No one can definitely prophesy when such serious organic involvement may occur. Some patients will go with what appears to be an apparent benign form of hypertension for many years, even without the more common symptoms of the disease, and then suddenly and for no known reason develop rapidly severe changes in the heart or kidneys or suffer a cerebral hemorrhage. The more common course is for the disease to progress slowly, the symptoms gradually increasing in number and severity and within a few years cardiac, renal or cerebral involvement becomes manifest. A few patients present a fulminating type of the disease called malignant hypertension with death ensuing in a few months.

The manifestations of hypertension are so many and so varied and the actual progress of the disease so uncertain, embracing a course from the extreme of a fatal termination in a few months to a seemingly benign course for many years,

that a true evaluation of any form of therapy is extremely difficult. This probably accounts for the very large number of therapeutic measures that have been advocated. Very thorough and honest appraisals have been made of all the usual drugs recommended for the treatment of hypertension and it has been unequivocably demonstrated that they are of no practical value.

It is true that a number of drugs will very temporarily reduce the blood pressure but such temporary reduction is of no value in the treatment of a chronic disease. Nitroglycerine is of course indicated in the emergency of angina pectoris but only for its immediate and extremely temporary effect. It is valueless in preventing future attacks. The only drug which today is recommended by some of our leading medical men is potassium thiocyanate. This must be given with a great deal of caution and frequent blood level determinations obtained. Even with the most careful precautions and when the level is maintained at a theoretical safe concentration, toxic symptoms sometime develop; and it is because of this uncertainty, both in its toxicity and in its therapeutic benefit, that many leading internists do not recommend it. It is therefore fortunate that surgery offers worth while improvement in a significant percentage of cases..

The cause of hypertension is unknown but an ever increasing mass of data indicates that heredity plays an important rôle. The vast majority of cases give a definite family history of hypertension. In fact it is rather unusual to get a history of a patient neither of whose parents had an abnormally high blood pressure. Commonly, in addition to either the father or the mother having had hypertension, we obtain a history that one or more brothers or sisters of that particular parent also had an elevated blood pressure. In some families there is a very definite history of several dying from cerebrovascular accidents. In another family we will have renal complications as the cause of death. In a few we

have been able to trace the hypertension for three generations. With a hypertensive family history we can be quite certain that one or more of the children will have hypertension and the percentage greatly increases if both parents are hypertensive.

Frequently the disease appears at a younger age in the children than in the parents. One young woman who came to us for a splanchnicectomy to relieve her hypertension gave the following striking family history: Her mother had died of cardiorenal complications from hypertension, her mother's sister likewise had hypertension and died of cardiorenal disease. The patient had five sisters, she being the youngest in the family; all five sisters had had hypertension and had died before the patient came to us. We have had numerous other family histories nearly as striking. Certainly with such evidence the rôle of heredity must be given very serious consideration. We do not believe that it is an abnormality in the autonomic nervous system which is inherited. This variation from the normal is manifested by excessive responses to normal stimuli. In other words, we believe that there is a congenital tendency of the autonomic system to over-react and to respond to a given stimulus by either greater or more prolonged vasoconstriction, which in the hypertensive involves the renal circulation.

The development of hypertension is usually insidious and many times an early hypertensive case is discovered largely by accident. The routine physical examinations required by many industrial firms for all applicants at the time of employment frequently disclose some elevation in blood pressure in young individuals who are completely symptom-free. A surprisingly large number of early hypertensive cases was found during the induction examination for the Army and Navy. Life insurance examinations have been a fruitful source in the discovery of hypertension. In spite of these various opportunities for the detection of the disease the great majority of patients do not know that they have an abnormally elevated blood pressure until they consult a physician because of severe symptoms such as incapacitating headaches and abnormal nervousness and irritability, or because of actual organic changes in the heart or kidneys. It is therefore impossible in the large proportion of our patients to make a definite statement of how long hypertension has actually existed.

Certain precipitating factors seem at times to be the immediate cause of hypertension; particularly is this true of pregnancy. Rather commonly the history discloses that hypertension first developed during pregnancy. Sometimes the blood pressure drops to normal after delivery at full term only to rise again and generally to a higher level at a subsequent pregnancy. In such cases the blood pressure level seldom comes down after termination of that pregnancy. We are of the opinion that the pregnancy simply precipitates the disease in a woman who, in all probability, would have developed hypertension at some time later. In other words she was a potential hypertensive individual and it took only this added change in her physiology to bring on the hypertension at an earlier date.

Unfortunately, it is impossible to prove or disprove this assertion. The toxemia of pregnancy appears to be much more common in women who do have elevated blood pressure and it has been shown that roughly 25 per cent of women who have had such a toxemia have a persistent hypertension postpartum.[3] The magnitude of this factor in the over-all picture of hypertension is shown by the following figures: toxemia occurs in about 8 per cent of all pregnancies, therefore, 2 per cent of all women who become pregnant will be expected to develop a hypertension which persists.[19] At the present rate of births in this country this would give a figure of more than 70,000 women each year who can be expected to develop hypertension as the result of pregnancy.[10]

Prolonged emotional strain such as worry over financial matters or domestic problems is frequently given as a precipitating cause of hypertension. Certainly in times of severe stress, as in war, financial crises or some other widespread causes for severe anxiety, the number of hypertensive patients seems to be greatly increased. There is also considerable evidence that very severe illnesses, such as pneumonia, mastoiditis and often such short illnesses as acute appendicitis, may be precipitating factors. However, such opinions are exceedingly difficult to prove since the majority of people do not have their blood pressures taken until real need for medical attention develops.

DIAGNOSIS

The diagnosis of arterial hypertension, commonly spoken of as essential hypertension and by some as diastolic hypertension, is primarily based on the findings of an elevated systolic and diastolic pressure. A single observation is of little significance. Repeated pressures must be taken under various circumstances. Several criteria to determine the hypertensive state are in use. Goldring and Chasis[5] make the following statement, "From three to sixteen years of age the systolic blood pressure rises progressively from 90 mm. Hg to 115 mm. Hg and the diastolic from 65 mm. Hg to 75 mm. Hg; at forty years of age the upper limit is 140 mm. Hg in the systolic and 90 mm. Hg in the diastolic phase; above forty the upper normal limit is 150 mm. Hg in the systolic and 90 mm. Hg in the diastolic phase." Some authors are more extreme and consider a diastolic reading of over 80 mm. abnormal at any age. However, the majority of medical men consider the diastolic as normal if below 100 mm. at age forty or older. The method of taking the blood pressure accounts for many discrepancies. Usually the blood pressure is different in the reclining, sitting and standing positions. It may differ considerably in the two arms. The emotional status of the patient at the time of examination is an important factor and must be considered in each case. A patient manifesting nervousness over the examination or anxiety over some other matter will have a pressure definitely above his usual level. I saw this demonstrated many times during the war when young men who were particularly desirous of getting into the airforce were refused because of a slightly elevated blood pressure. On numerous examinations, their blood pressures were found to be absolutely normal by their home physicians and by myself. Almost routinely the blood pressure will be found at a lower level, both in normal and in hypertensive patients, if the reading is taken following a short rest instead of being taken immediately upon entering the examining room. It is believed by some that a pre-hypertensive stage can be demonstrated, but there is much conflicting evidence in this matter and at present we have no test which will definitely indicate whether a certain individual will or will not subsequently develop hypertension. However, certain leads have been established and it can be stated quite definitely that the individuals who maintain a relatively stable normal blood pressure level under a variety of conditions and at different times will probably not develop hypertension. Only about 3 per cent of such apparently stable individuals have subsequently been found to have hypertension. Conversely, 70 per cent of apparently normal individuals who showed temporary elevations in systolic and diastolic pressures at various times subsequently developed hypertension.

The blood pressure should be taken initially in the horizontal, sitting and standing positions and in each arm. If an abnormally high level is recorded, the patient should rest for at least fifteen minutes, preferably longer, in a horizontal position and the blood pressure readings again recorded. Useful information is sometimes obtained by having this type of patient exercise such as running up and down a flight of stairs, recording the blood pressure

changes from such a standard type of exertion. As a matter of routine we always take three blood pressure readings in quick succession in each arm in each position of the body. If considerable variation is found on these three readings, subsequent readings are taken after a few minutes. It is possible to take direct arterial pressures by using a needle placed into the lumen of an artery, but for all practical purposes the mercury manometer or an accurate aneroid manometer with auscultatory determination of the pulse sounds at the elbow is satisfactory. It is desirable in all young people to determine the pulse pressure in the femoral artery since this will determine the presence or absence of coarctation of the aorta as a possible cause of the hypertension.

SYMPTOMS

Certain symptoms are so regularly complained of by hypertensive patients that one can be almost certain of the diagnosis, in many cases, before having any knowledge of the actual blood pressure level. However, in the very early case, symptoms are usually absent; even in advanced hypertension, symptoms may be entirely wanting. There does not seem to be any correlation between the height of blood pressure and symptoms. Some patients with a systolic blood pressure of 190 will complain of excrutiating suboccipital headaches; other patients with pressures ranging from 240 to 260 or even higher may never have a headache. However, it is generally noted in patients who do have symptoms that these are aggravated when the pressure is at an unusually high level.

The common symptoms of which a large number of patients complain are: headaches, especially suboccipital, nervousness, irritability, insomnia, nocturia, shortness of breath and ease of fatigue. The headaches are extremely common on awakening in the morning and disappear after the patient is up and about. In the more advanced cases the headaches may awaken the patient very early, sometimes at three or four in the morning, and it is impossible for them to get back to sleep even after taking the usual headache remedies. The headache may be bifrontal or bitemporal or extend over the entire head but most commonly it is in the back of the neck and suboccipital region and from there at times extends forward to the top of the head and frontal area. Severe suboccipital headaches occurring more or less regularly every morning are so characteristic of hypertension that when such a history is elicited I always expect to find a very material elevation in blood pressure. Some patients have a history of such headaches for many weeks at a time; others will complain of having them only one or two mornings a week. There may be periods, especially when the patient is on vacation or not under stress, when the headaches may entirely disappear. Frequently the taking of food and moving about will give relief but in some cases the headaches persist throughout the day. They may also appear at any time during the day especially if the patient becomes excited or is under stress or physically fatigued. It is rather a common medical practice to advise a patient to lie down in the afternoon and rest but many patients will state to do so will again bring on a suboccipital headache. At times these headaches are so severe as to be completely incapacitating. Generally there is no nausea or vomiting associated with them. Some patients will have complete relief of early morning headaches by sleeping with the head of the bed elevated. This is most easily accomplished by putting blocks of wood under the head of the bed raising it to a height of about 12 inches. In extreme cases the patient refuses to go to bed and sleeps sitting up in a chair.

Increasing nervousness is an exceedingly common complaint although some patients are not aware of this themselves and the family have to give the definite history. Not infrequently patients will state that they have always been rather nervous but never to the extent that they have felt since the onset of hypertension.

Frequently associated with increasing nervousness is irritability. This may be a comparatively minor complaint but in many patients it assumes really serious proportions. It may become so pronounced as to interfere with his business and with his domestic happiness. I have frequently had a patient make the following statement to me, "I love my children as much as any father possibly could, but it has gotten so now that anything they do, no matter how trivial, irritates me seriously." The nervousness and irritability are not dependent upon the height of the blood pressure. Some patients having this complaint have only moderately elevated pressures, while others who may have a very marked hypertension are relatively calm. However, in the majority of cases the nervousness and irritability are greater during periods in which the pressure is unusually high. A vicious circle certainly exists, the increased pressure making the patient more nervous and more irritable and this conversely raising the pressure still higher.

Nearly all patients complain of nocturia, generally once or twice at night, but in an appreciable number of cases nocturia is a very real problem and seriously interferes with sleep. A history of having to urinate every hour during the night is not especially uncommon. This is particularly found in those individuals whose concentrating ability is seriously impaired. Nocturnal dyspnea fortunately is a rather uncommon symptom since it indicates very serious cardiac damage. Very mild nocturnal dyspnea may be completely relieved by sleeping with the head on several pillows, but in the more severe cases the patient is unable to sleep in bed and has to sit in a nearly upright position in a chair Weight reduction, if the patient is obese, and digtalis may be of great value in relieving this distressing symptom but the over-all picture will always remain grave. Ease of fatigue is an exceedingly common complaint in hypertensive patients. It may be due somewhat to increased nervousness but seems more related to so-called

vital capacity. It does not have a definite relationship with the height of the blood pressure but may have a relationship with the cardiac and renal status. Some patients present just the opposite picture and with the development of hypertension become more and more dynamic and tireless. They feel keyed-up and under tension and do everything quickly. This is the type of personality so commonly considered hypertensive. The complaint of feeling always keyed-up and under tension is not confined, however, to this group and is, in fact, an exceedingly common complaint of all hypertensive patients. They frequently state that they cannot relax and this in itself makes sleep difficult.

Shortness of breath on mild physical exertion such as going up one flight of stairs is an extremely common complaint. It may or may not indicate cardiac damage. However, if the symptom is very definitely progressive and the patient is not unduly overweight, cardiac disturbance can be expected. Palpitation, either under exertion or while quiet, is also a very common complaint. It does not necessarily signify any organic change in the heart. In fact it is much more frequently associated with nervousness. Angina pectoris, either in a mild or severe form, occurs commonly in hypertensive patients. It may be the result of simple coronary spasm but in many it is probably due to actual arteriosclerosis of the coronary vessels. Particularly is this true when the hypertension is of long duration. Patients with angina pectoris are advised to stop smoking and to limit their activities and emotional strain. Nitroglycerine is invaluable as a relief agent at the onset of an attack. The patient with frequent anginal attacks has certainly a much graver prognosis. Fortunately many patients with angina have complete relief following splanchnicectomy. This suggests that vascular spasm rather than arteriosclerosis was the immediate factor. Edema of the ankles when occurring regularly is of serious import since it is most frequently associated

with cardiac decompensation. Other causes such as varicose veins should of course be ruled out.

Nausea and vomiting are uncommon symptoms of hypertension even in the patients who suffer from severe headaches. A history of repeated attacks of nausea and vomiting is highly suggestive of the so-called malignant type of hypertension. In most patients with these symptoms evidences of severe renal damage will be found.

Mental symptoms of a more or less severe degree occur very commonly in hypertensive patients. As a rule they are slight and characterized chiefly by difficulty in concentration and a slightly impaired memory, especially for recent matters of little consequence. However, many patients complain that "their head feels thick," that they cannot think as they formerly did, that at times they seem to be in a fog; some complain that their loss of memory is a serious handicap in their business. Some patients have apparently not been cognizant of any previous mental difficulty until after a splanchnicectomy; then they commonly remark that they can now think clearly, that they can concentrate and they often state, "it seems as though a fog had been wiped away." From these mild evidences of hypertensive encephalopathy we have various grades in the individual who is entirely incapable of carrying on any active mental processes. Usually the mental changes are insidious and their development very gradual. Even marked mental deficiency may be found in patients who apparently have not had a cerebral accident, either cerebral hemorrhage or thrombosis. All the signs and symptoms of hypertensive encephalopathy are greatly increased when a so-called cerebral accident occurs. Small intracranial hemorrhages can occur without danger to life but I believe the majority of so-called strokes which the patient survives are actually due to vascular thrombosis. I do not deny the possibility of cerebral vascular spasm but believe, at

least in the majority of cases, that even the transitory symptoms of a stroke are due to small thrombii.

Blurring of the vision, either recurrent and transitory, or of more prolonged periods is a rather common symptom in the more advanced stages of hypertensive disease. At times the blurring lasts only for a few minutes and strongly suggests angiospasm. More prolonged periods of blurring in one eye or in both are most commonly associated with retinal hemorrhages. As a rule such hemorrhages are associated with a rather marked hypertension but may occur in individuals whose pressure is not over 170 systolic. It is also true that in some individuals who have exceedingly high blood pressures, the fundoscopic examination never discloses a hemorrhage. Sudden complete loss of vision in one eye generally indicates either a massive retinal hemorrhage or a thrombosis of a central artery or vein or one of their main branches. A massive retinal hemorrhage may be slowly absorbed and useful vision finally restored, but a thrombosis of the central artery results in complete, permanent loss of vision. Impaired vision which fluctuates to some extent may be due to repeated small retinal hemorrhages and exudates but as a rule these do not impinge upon the macula and rather extensive hemorrhages are often seen without noticeable impairment of vision. Severe bilateral impairment of vision is most often due to a high degree of papilledema with the usually associated extensive hemorrhages and exudates. The finding of papilledema indicates a fulminating type of hypertension usually designated as malignant hypertension. The progress of the disease in these cases is so rapid and the prognosis so poor that many consider malignant hypertension as a separate disease entity.

Other hypertensive symptoms rather commonly complained of but which are not indicative of any disease picture are dizziness, vertigo, tinnitus and mild deafness. Many hypertensive patients com-

plain of transitory dizziness, sometimes associated with severe suboccipital headaches, at other times only present upon sudden changing of position. True vertigo is quite rare. Many patients complain of tinnitus, generally in both ears, and of a rather mild degree. Much more frequently patients complain of feeling the heart beat in the ear especially when reclining. Mild deafness certainly can be on a hypertensive basis since careful audiometer examinations prior and subsequent to splanchnicectomy have shown definite improvement in the hearing following operation.

TREATMENT

The commonly accepted drugs for the treatment of hypertension have been proved valueless. Certain general measures, however, are indicated, especially in the early or mild hypertensive patients. Reduction in weight is important in all obese patients. Occasionally reduction to a normal weight will materially lower the hypertension. In all cases it takes an added strain from the heart. When surgical therapy is advised, weight reduction is definitely indicated since the operation is much easier of accomplishment and the patient's subsequent care and comfort is enhanced. Of course, not all patients with hypertension are overweight; some in fact are below normal. In such individuals supplementary feedings, especially of a rather high caloric diet, are indicated; and if the appetite is poor, multivitamins may do much to improve it. Adequate rest at night is important and whenever possible a rest period after the noon meal should be taken. Few patients can afford to follow the advice so commonly given, namely, to give up all their work and to stop worrying about their condition. Worry is a recognized factor in heightening the blood pressure, but it is an impossibility for the average person to have to give up a gainful occupation and not worry about doing so.

Many special diets for the treatment of hypertension have been recommended but with two exceptions they are probably of little value. The exceptions are the rice diet[8] and the very low sodium diet.[1] Some patients adherring strictly to the rice diet have shown rather remarkable lowering of blood pressure and amelioration of their symptoms. However, on returning to a regular diet the evidences of hypertension quite promptly return. A low sodium diet, that is one containing only 200 mg. of sodium per day, has been found equally efficacious; some patients have a reduction in symptoms and in a few cases some reduction in blood pressure. If kidney function is adequate, radical reduction in protein intake is not indicated. However, if the blood non-protein nitrogen is rather markedly elevated, the diet should be low in protein. For weight reduction we generally recommend a 1,200 calorie diet; but in very obese patients or in those in whom weight reduction preparatory to a greatly needed operation must be accomplished rapidly, we have prescribed a diet of only 800 calories, supplementing this, of course, with vitamins. On a few occasions a diet as low as 350 calories has been prescribed.

Potassium thiocyanate has been highly recommended by many clinicians. It must be given with caution and the blood level determined at weekly intervals. It is generally considered that a level of at least 6 mg. per cent is necessary to have any therapeutic value and the level should not go above 12 mg. per cent. However, some patients show severe toxic reactions at levels even below 6 mg. per cent and for this reason many clinicians refuse to use this drug. It is a palliative measure at best. The symptoms may be alleviated or actually disappear and in some cases the blood pressure shows satisfactory reduction, but as soon as the drug is discontinued the hypertensive symptoms and elevated blood pressure return.

We believe that surgery offers the best form of therapy and a sufficient number of patients have now been operated upon and studied for a sufficiently long period

to make definite statements as to the value of splanchnicectomy. We do not advocate surgery for all patients. A very mild hypertensive patient may live for many years without apparent organic damage. However, if there are evidences of a progression of the disease, surgery seems definitely indicated. Far too often the medical man watches the progress of the disease until irreparable damage has resulted, either in the brain, the heart or the kidneys and then refers the patient for surgery. This is neither fair to the paitient, the surgeon nor to the procedure which he advocates.

RATIONALE FOR SURGICAL TREATMENT

The rationale for the surgical treatment of hypertension is based upon our concept of the disease, particularly of its etiology. Although many theories have been advanced, no one yet has actually been able to establish the true, primary cause of arterial hypertension. We have accepted the renal-humeral theory, first satisfactorily demonstrated by Goldblatt,[4] who showed conclusively that renal ischemia, or at least the interference with the renal hemodynamic state, produced a continuous elevation of blood pressure without at first any evidence of impaired renal excretion. It has been shown by numerous workers that such interference with the blood supply of the kidneys results in an excessive amount of a renal pseudoglobulin called rennin being liberated into the renal veins. This rennin reacting with another pseudoglobulin probably arising in the liver and named preangitonin (rennin-activator of Page) forms an active vasopressor substance called angiotonin. This latter substance causes arteriolar constriction of sufficient degree to raise peripheral resistance throughout the body with a consequent elevation in blood pressure. Goldring and Chasis[5] make the following statement, "Hypertension is the sign representing the alteration in hemodynamics, and hypertensive disease is a clinical entity in which an unknown pressor mechanism initiates arteriolar vaso-con-

striction, elevated blood pressure, and vascular sequelae." We believe this vasopressor substance is angiotonin. The question naturally arises, What is the cause of this change in renal hemodynamics? There are probably numerous causes but we believe in the majority of cases it is of neurogenic origin.

The recent brilliant experimental work of Trueta, Barclay, Franklin, Daniel and Prichard[4] has clearly demonstrated that the splanchnic nerves can be reflexly stimulated with resulting severe renal ischemia. They have shown, for example, that stimulation of the sciatic nerve can reflexly stimulate the splanchnic nerves with a resulting vasoconstriction in the arterioles of the renal cortex so that the blood flow is partially or even completely abolished through this portion of the kidney. Direct stimulation of the distal end of the cut splanchnic nerve gave the same results. It had previously been shown that electrical stimulation of the splanchnic nerves in dogs elevated the blood pressure, probably by constricting the arterioles in the kidney, resulting in renal ischemia.

It is our opinion that in human hypertension an abnormal stimulation of the splanchnic nerves originates in the autonomic centers of the brain, resulting in similar changes in the renal hemodynamics. The activating stimulus may be psychic, reflexes from stimuli originating in other parts of the body, or it may be chemical in nature. Normally stimuli are constantly influencing the sympathetic nervous system but in certain diseases these stimuli result in excessive responses. This is seen for example in Raynaud's disease. We believe that the abnormal excessive response to normal stimuli, sufficient to cause renal ischemia, is based on a congenital abnormality in the autonomic nervous system whereby excessive responses are evoked by the normal stimuli reaching those centers. It is our opinnion that this tendency of the autonomic system to overreact, to respond to a given stimulus by either greater or more pro-

longed vasoconstriction of the renal vessels, is inherited. The family history of most hypertensive patients lends weight to this theory. If our theory is correct and there is a large amount of evidence to support it, splanchnicectomy if performed before permanent changes occur in the blood vessels should improve the renal hemodynamic state.

SELECTION OF PATIENTS FOR SPLANCHNICECTOMY

We believe that any patient showing a progressive type of hypertension and whose age and cardiac and renal status is satisfactory should have the benefit of surgical intervention. Age certainly is a factor. The younger group have a much higher percentage of worth while results than the older age groups. However, so many factors determine the ultimate result of the operation that each individual case should be considered on its own merits. We have more or less arbitrarily, fixed an age limit of fifty-three. When we originated the procedure of bilateral supradiaphragmatic splanchnicectomy for the treatment of hypertension fourteen years ago, we set an arbitrary age limit of forty-five; but it was soon found that a rather high percentage of patients at that age frequently responded very well and so the age limit was gradually extended, still with gratifying results. We may still further extend it. At present we seldom operate on a patient age fifty-four or above, unless that patient has incapacitating symptoms or shows evidence of a very rapidly progressive type of hypertension such as the so-called malignant phase. The youngest patient we have operated upon was aged eight, the oldest sixty-three.

The question frequently arises, Should a patient who has had a cerebral accident be operated upon for the treatment of his hypertension? We realize that a cerebral accident presupposes abnormal pathologic changes in the cerebral vessels and that the risk and the prognosis of such a patient is influenced thereby. Under no circum-

stances would we operate upon a patient shortly after a cerebral hemorrhage or thrombosis; but if the patient has made a complete recovery both physically and mentally and in addition shows a progressive type of hypertension, we would advise operation. However, if there is evidence that the blood pressure has been remaining at the same level for a prolonged period and the patient has had no increase in symptoms or further impairment of renal or cardiac condition and especially if the patient is over fifty, we would probably advise against splanchnicectomy.

Cardiac decompensation is, of course, an absolute contraindication. However, some patients who have given a history of noctural dyspnea, ankle edema and excessive shortness of breath on mild exertion but who have responded satisfactorily to digitalis and bed rest may be operated upon with a comparatively low operative mortality and definite hopes of at least temporary improvement. It should be pointed out that the majority of deaths occurring in the first five years postoperatively have been among those patients who had had such evidences of cardiac decompensation before operation.[5] Deaths within this period have occurred in patients whose blood pressure had been reduced and maintained at a normal level up to the time of death, two, three or more years following operation.

Renal insufficiency as measured by the level of the blood non-protein nitrogen and blood urea is also a definite contraindication to operation. We have found that only in exceptional cases have the patients had more than temporary improvement if the blood non-protein nitrogen, when the patient had been on a large fluid intake, was above 45 mg. per cent. Such temporary improvement might be for a year or more and possibly could be considered a justification for the operation. However, we generally do not advise operation if the non-protein nitrogen is above 45 with the single exception of those patients with malignant hypertension. There we realize that a defi-

nite emergency exists and we are willing to operate when the non-protein nitrogen cannot be brought below 56 mg. per cent.

The duration of hypertension probably is a factor in the ultimate outcome but unfortunately it is only an exceptional case in which the actual duration is known. We therefore do not have sufficient data upon which to base a positive statement. The various tests such as intravenous pentothal, sodium amytal, spinal anesthesia, cold pressor tests and so forth, which have been used so extensively in the hopes of finding some criteria which would definitely indicate the probable result of the operation, have proved valueless. It is true that some of these tests may show a general trend which statistically is of interest, but in the determination of the probable outcome of any individual they need not be considered.

Malignant hypertension is a definite indication for splanchnicectomy rather than a contraindication as some believe. It is the only form of therapy offering any chance for improvement in this type of hypertension. The prognosis is so absolutely hopeless without operation and the results of splanchnicectomy in some cases have been so brilliant that we advise operation even when the blood non-protein nitrogen is at a higher level than would be acceptable for the less serious case.

Certain tests are routinely made on all hypertensive patients since it is necessary to have full data on the cardiac and renal status before making a decision regarding a splanchnicectomy. The fundoscopic examination is also important since it gives us valuable information regarding the type of hypertension and its probable rate of progress. Full preoperative studies with similar postoperative studies over a period of years are essential if we are to evaluate properly the results of surgery.

The ophthalmologic examination is principally of the fundus although the visual acuity is recorded and in certain cases visual fields are made. The fundoscopic examination should be made with the pupil well dilated. The condition of the arteries and veins and the presence of angiospasm and the degree of arteriosclerosis is noted. The recognition of angiospasm as distinct from arteriosclerotic changes requires much practice. It is generally believed that the presence of angiospasm denotes a progressive type of hypertension. In the early stages of the disease the only pathological condition noted may be an increase in the reflex stripe of the arteries and arteriovenous nicking. Hemorrhages both striate and flame-shaped indicate a more serious phase of hypertension. The presence of cotton wool exudates, with or without associated hemorrhage, also indicates rather advanced hypertension. Edema of the borders of the discs should be carefully looked for and the actual presence of papilledema of 1 diopter or more is considered pathognomonic of malignant hypertension.

The cardiac status is determined by an electrocardiogram, an orthodiagram and a teleoroentgenogram. If there is evidence of coronary involvement, an additional electrocardiogram with chest leads is made. In some special studies ballistocardiographic records have also been made. The data from the above findings are of course correlated with the clinical history, with particular attention to shortness of breath on exertion, nocturnal dyspnea, ankle edema and attacks of angina pectoris.

The renal function is determined by complete urinalysis, urea clearance, water concentration, blood non-protein nitrogen and blood urea, and intravenous pyelograms. Formerly we used a thirty-six-hour water concentration test but this proved extremely burdensome for many of the patients and in recent years we have compromised by using an eighteen-hour test made in the following way. The patient has his usual meal at 6 P.M., then no food or fluids of any kind are taken until afternoon of the following day. A urine sample is obtained at 8 A.M. again at 10 A.M. and again at 12 noon, and the specific gravity and presence and amount, if present, of albumin separately determined. This test has given somewhat lower values than we

obtained on the thirty-six hour test. The blood non-protein nitrogen and blood urea determinations should be made when the patient is on a large fluid intake. Intravenous pyelograms have been made on all patients to rule out gross pathologic disorder in either kidney. Retrograde pyelograms are necessary when the intravenous pyelograms do not give unequivocal data. Considerable discussion has arisen over the requirement of an intravenous pyelogram since it is comparatively rare to find a gross pathologic condition sufficient to be a possible cause of the hypertension. Possibly it is only necessary to make this test in those individuals who give a suggestive history of severe renal complications and in children in whom a possible congenital renal lesion may be present. The presence of bilateral chronically contracted kidneys is a definite contraindication to operation, so too are polycystic kidneys. When one kidney is found by x-ray to be diseased and the other normal, a nephrectomy has been advised but in only a few cases has this materially improved the hypertension. In many cases the subsequent bilateral splanchnicectomy has given excellent results. Craig,[2] who performs his splanchnicectomies in two stages, advocates a unilateral splanchnicectomy on the side of the diseased kidney at the time of nephrectomy; then later if satisfactory results have not been obtained, the second stage operation for splanchnicectomy on the opposite side is performed.

The use of tetra-ethyl-ammonium as a test for operative prognosis has been made in a rather large series of cases. Like other tests involving generalized vasodilation, it has been found of no definite value in the prognosis of any single individual; however, certain generalities can be made.[9] In a large series it is demonstrated that those patients who respond with a significant drop in blood pressure with the administration of tetra-ethyl-ammonium intravenously will show a high percentage of good results following splanchnicectomy, and conversely the group who do not respond to tetra-ethyl-ammonium have a low percentage of satisfactory lowering of the blood pressure following surgery. However, the test in no way indicates whether the patient will receive worth while symptomatic relief. It has been repeatedly demonstrated that the relief of distressing symptoms is not closely correlated with the reduction of blood pressure. Tetra-ethyl-ammonium should not be used as a therapeutic agent but simply as a test of the response to a vaso-dilating drug to help determine the possible result of operation.

Many hypertensive patients show evidences of encephalopathies varying in degree from slight loss of memory or difficulty in concentrating to marked mental clouding. We are now making electroencephalograms using an 8 channel machine on all such patients. Similar recordings will be made at suitable postoperative periods. We hope with these data to be able to determine how much of the hypertensive encephalopathy is due to actual organic changes and how much improvement in the mental status, following splanchnicectomy, may be expected.

SURGICAL PROCEDURES

The surgical technic for the treatment of hypertension varies in different clinics, chiefly in the extent of the sympathetic denervation. Since our first operation[12] for hypertension in November, 1933, we have routinely carried out a bilateral supradiaphragmatic splanchnicectomy.[13] In some clinics,[5-12] this is combined with resection of the first or first and second lumbar ganglia subdiaphragmatically. Other variations in the procedure extend the excision of the thoracic ganglia up to include the third and in one clinic the first thoracic ganglion.[5]

The operation of bilateral supradiaphragmatic splanchnicectomy has now been performed in our clinic on about 2,000 patients over a fourteen-year-period. It consists of the bilateral resection of a very long segment of each greater splanchnic nerve and of the lower thoracic sympa-

thetic ganglia with the lesser and least splanchnic nerves arising from them. In our earlier cases we resected about 10 to 12 cm. of each greater splanchnic nerve and excised the tenth, eleventh and twelfth sympathetic ganglia bilaterally. With added experience, the addition of special instruments and a new form of lighted retractor we have been able to resect routinely 20 cm. or more of the greater splanchnic nerve and the seventh to the twelfth inclusive sympathetic ganglia. In some cases it is possible to resect the sixth and even the fifth thoracic ganglia through the same approach used formerly, namely, resection of a portion of the eleventh rib. Avertin anesthesia, using 110 mg. per Kg. of body weight, supplemented with nitrous oxide and oxygen, has been the anesthetic of choice. Intravenous physiologic glucose solution is given slowly throughout the operation.

Two scratch marks outlining the bilateral incisions are made, each about 10 cm. long on thin individuals, longer in heavily built or fat patients. These incisions are parallel in vertical direction, about 8 cm. apart and center over the eleventh intercostal spaces. The incision is carried through the skin, superficial fat and lumbodorsal fascia. The longissimus dorsi is retracted medially and the eleventh rib exposed. The intercostal muscles are separated from the rib as is the pleura and a segment 5 or 6 cm. long of the rib resected. The excision extends nearly to the head of the rib. A small piece of the intercostal muscle is removed and saved for biopsy examination to disclose pathologic changes in the small arterioles. The intercostal artery and vein lying just above the twelfth rib are double clipped and a segment about 5 cm. long excised. Care is taken not to damage the adjacent intercostal nerve. The pleura is then very carefully separated by semi-sharp dissection from the bodies of the vertebrae and this dissection is carried medially to the level of the anterior border of the vertebral bodies. The pleura is separated caudally to the crus of the

diaphragm and cephalad as far as it can be conveniently exposed, usually to a level above the seventh thoracic ganglion. In some individuals the configuration of the chest is such that this dissection can be carried up above the fifth thoracic vertebra without difficulty. On the right side the greater splanchnic nerve lies between the pleura and the inferior border of the vertebrae; on the left it lies on top of the aorta which in most hypertensive cases is displaced laterally. The greater splanchnic nerve is grasped with long forceps and upward traction made. Frequently such traction pulls the upper pole of the celiac ganglion through the opening in the diaphragm for the passage of the greater splanchnic nerve. The nerve is then dessicated with an electric current as it enters the ganglion and divided. The distal stump retracts below the diaphragm. The nerve is then carefully dissected from its bed of fascia and freed upward to the level of the sixth or seventh vertebra.

The thoracic ganglionated chain lies more dorsally on the bodies of the vertebra. The ramus from the eleventh intercostal nerve to the eleventh thoracic ganglion is dessicated, divided, and then the chain dissected downward exposing the twelfth ramus and ganglion. In many cases, because of rather high reflection of the diaphragm, it is necessary to divide some of the fibers of the diaphragm at their attachment to the vertebrae until the twelfth ganglion is fully exposed. This may even lie on the peritoneal side of the diaphragm. The chain below the twelfth is dessicated and divided and the least splanchnic nerve avulsed as the sympathetic chain is pulled upward. Resection is then carried upward with a special semi-sharp elevator and in turn the rami to the tenth, ninth, eighth, seventh and some times the sixth, rarely the fifth, ganglia are dessicated and cut and then the chain above the uppermost exposed ganglia is dessicated with the electric current and divided. As a rule the main trunk of the greater splanchnic nerve will be found arising from the uppermost of the

exposed thoracic ganglia. The entire chain and the attached greater splanchnic nerve are removed from the extrapleural space. The various lesser splanchnic nerves are either avulsed or, if they are especially strong, dessicated and divided at the level of the diaphragm. All bleeding points are carefully dessicated. The extrapleural space is then filled with Ringer's solution and the lung fully expanded by increasing the pressure in the rebreathing bag of the anesthetic machine.

If a hole is inadvertently torn in the pleura during any stage of the operation, it in no way modifies the procedure and the operation is continued without difficulty. If the pleura has been accidentally torn on the right side, we do not hesitate to proceed with the left splanchnicectomy. Of course special care is taken to assure full lung expansion before closure of the incision. Rarely is there suction into the pleural cavity during closure and in such cases aspiration through a needle at the completion of operation may be necessary. The reason for filling the extrapleural space with Ringer's solution before expanding the lung is to assure no air being trapped in the mediastinum which would of itself cause no serious complication but may result in some crepitation in the neck and be a cause of worry to the patient. Any Ringer's solution not forced out by the expansion of the lung is of course readily absorbed. The incision is closed in layers and immediately a similar procedure carried out on the left side. The combined bilateral operation takes from forty-five minutes to one and one-half hours. Frequent blood pressure recordings are made throughout the operation. If the blood pressure drops below 120 mm. Hg systolic, supportive measures are used. We have found neosynephrin very useful in maintaining the blood pressure at a safe level.

The blood pressure may drop at any time during the next few hours and therefore the patient is kept flat in bed with the foot of the bed elevated for the first twelve hours. It may be necessary to give neo-

synephrin on one or two occasions during the first few hours. Patients are encouraged to sit up at an early date and those who feel able to be out of bed may get up within a day or two following operation. On first assuming the upright position there may be a postural hypotension. This as a rule corrects itself within a few days. It has never been necessary to bandage the legs or put compression upon the abdomen. However, we always advise the patients first to assume the upright position for a few minutes, then to get out of bed slowly. Most patients leave the hospital on the twelfth postoperative day and it is rather unusual for patients to have to remain in more than fourteen days. We advise a rest period at home of at least four weeks before resuming any active duties.

Few complications have arisen in about 2,000 operations. Five patients have had an injury to the thoracic duct and have developed extrapleural collections of chyle. Three of these were successfully treated by repeated aspiration for a few days. The other two required operation; in one the leaking thoracic duct was found damaged at the crus of the diaphragm; in the other a small radical lying on top of and to the outer side of the aorta about the level of the tenth interspace had apparently been torn. A clip upon this radical stopped further leakage of chyle and convalescence was uninterrupted.

Occasionally a mild atelectatic condition has developed. Usually this has been controlled by insisting upon the patient coughing but on a few occasions it has been necessary to bronchoscope the patient and aspirate the mucous plug. Pneumonia has not been a complication for several years. Previously, when atelectasis had not been recognized, a few patients developed mild bronchial pneumonia. Because we insist upon the patient moving about freely and encourage early ambulation, thrombophlebitis of the lower extremities has not occurred.

A few cases of coronary thrombosis, either during the operation, immediately

after or before discharge from the hospital, have occurred, but in none who did not have a previous history of coronary disease. A few individuals have had cerebral thrombosis, apparently as a result of a marked drop in blood pressure occurring during or immediately after the operation. These were all in individuals with considerable evidence of cerebral arteriosclerosis. It is for this reason that we are exceedingly careful to keep up the blood pressure to at least 120 mm. Hg systolic throughout the operation and during early convalescence.

SUMMARY OF INDICATIONS AND CONTRAINDICATIONS FOR SPLANCHNICECTOMY

Our criteria for operation may be summarized as follows: an age below fifty-four but with occasional exceptions; a more or less continuously elevated blood pressure with a systolic over 170 and a diastolic over 105; definite evidence in the milder hypertensive cases of a progression in the disease picture; a non-protein nitrogen below 45 mg. per cent, preferably below 40 mg. per cent; a compensated heart without recent history of coronary thrombosis. We prefer that the patient have a relatively normal cerebral function but have operated upon many showing rather marked hypertensive encephalopathy with gratifying results. Exceptions to the above general rules have been made, especially in operating upon patients who are older, generally because of incapacitating symptoms or evidences of a rapidly progressive type of hypertension. Only in the malignant type do we believe operation is worth while if the non-protein nitrogen is above 45 mg. per cent.

CLASSIFICATION OF HYPERTENSION

The manifestations of hypertension are so varied that it is necessary to have a workable classification in order to evaluate properly the results of surgical therapy. Even with a very complex classification so many factors are involved that a true statistical study is difficult. Arterial hypertension is a generalized disease, all organs of the body being affected, although the principal pathologic disorder may be greatest in one or two important organs. The commonly accepted classification is that of Wagener and Keith[22] but we did not find this entirely satisfactory and hence Isberg and I[15] enlarged the grouping from four to six. We suggest the following classification as very useful since it emphasizes the dominant pathologic picture:

Group 1. Early, mild hypertension. These patients are entirely asymptomatic, have normal or grade 1 fundi, and show no evidence of cardiac, renal or cerebral involvement.

Group 2. Symptoms predominate. All patients in this group complain of symptoms and have mild changes in the retinal blood vessels but display no evidences of cardiac, renal or cerebral impairment.

Group 3. Organic heart disease is predominant. In each case the diagnosis of heart disease is confirmed by either or both a definitely abnormal electrocardiogram and a teleoroentgenogram showing cardiac enlargement.

Group 4. Cerebrovascular disease is predominant. Each patient in this group has had one or more previous cerebral accidents.

Group 5. Impaired renal function is predominant. Each patient shows diminished concentrating ability and urea clearance values.

Group 6. Malignant hypertension. These patients have severe neuroretinitis with definite papilledema of one diopter or more and display a rapidly progressive, downhill course.

RESULTS OF BILATERAL SUPRADIAPHRAGMATIC SPLANCHNICECTOMY FOR HYPERTENSION

A bilateral supradiaphragmatic splanchnicectomy has been performed upon approximately 2,000 patients at the University Hospital over a period of fourteen years. A large proportion of these patients have

returned to the hospital for periodic examinations. Some have returned yearly; others only after a period of several years. Very complete postoperative studies have been made by the home physician in many cases when the patient could not come to us. We have attempted to obtain in each case complete postoperative studies, including a careful interval history, ocular fundoscopic examinations, water concentration, urea clearance, blood nonprotein nitrogen, electrocardiogram, orthodiagram and repeated blood pressure readings. Unfortunately it has not been possible to get complete studies on all the patients returning for check-up examinations. This accounts for the different numbers of patients included in the statistical studies on postoperative blood pressure levels and renal and cardiac status. We believe an interval of at least one year following operation should elapse before making any evaluation of the results of the procedure. Some of our statistics are based on studies from one to nearly fourteen years following operation. Special long term studies have been made at one postoperative period five to thirteen years.

SYMPTOMATIC RELIEF

This has been striking and in general is apparently only roughly correlated with the reduction in blood pressure. However, certain symptoms such as shortness of breath and nocturia are quite closely related with improvement in cardiac and renal function. The excruciating headaches which are usually suboccipital and the marked nervousness and irritability are in most cases completely relieved. Insomnia, nocturia and distressing palpitation of the heart have been completely or at least greatly improved in 86 per cent of the patients still living. This percentage of improvement has persisted almost unchanged for postoperative periods of from five to twelve years. Visual improvement has been striking especially in the malignant cases in which vision is often very seriously impaired. In every surviving pa-

tient who had had visual difficulties on a hypertensive basis the improvement has been practically 100 per cent. Some who were so blind that they could not distinguish faces have had a return to normal visual acuity, usually within a very few weeks.

BLOOD PRESSURE RESPONSE TO SPLANCHNICECTOMY

Blood pressure, both systolic and diastolic, was significantly reduced over a period of five to twelve years after operation in 81.3 per cent of the patients still living.[15] No improvement in blood pressure level was found in 12.7 per cent but over the course of these years only 6 per cent had had an increase in blood pressure. If we include in this statistical study those patients who had died one to twelve years postoperatively with the group still living five to twelve years after operation, we have definite improvement in the blood pressure in 46.7 per cent of the entire series. It should be borne in mind that 49 per cent of those that died in this period had malignant hypertension. We have used the following criteria for normal blood pressure readings: age twenty to forty years— 140/90; age forty to sixty years—150/95. While some disagree, these figures are generally accepted. Using this standard 20.3 per cent of our patients have maintained normal blood pressures following splanchnicectomy for five to twelve years. Those showing a worth while reduction to normal or approximately normal at the end of one year have, with very few exceptions, maintained the same blood pressure level at each subsequent examination, sometimes yearly, up to the longest postoperative period in our series, which is nearly fourteen years. A few cases have shown a return of the hypertension after maintaining normal levels from three to ten years postoperatively. It has been suggested that this signifies a regeneration of autonomic fibers. A marked improvement, but not to a normal level, based on a reduction of over 80 mm. Hg systolic

and 25 mm. Hg diastolic, has been maintained in 26 per cent of our patients for the entire postoperative period of five to twelve years. An additional 35 per cent have had what we call significant improvement, that is a maintained reduction in blood pressure of more than 40 mm. systolic and 15 diastolic. Combining the three groups, those with a reduction to normal, those with marked reduction, and those with significant reductions, we have an over-all improvement in blood pressure in 81.3 per cent of the living cases, which have been maintained from five to twelve years. If we consider the blood pressure of those who died before the end of this period, most of whom were malignant cases, we still have an improvement in blood pressure being maintained in nearly 50 per cent of the cases.

FUNDOSCOPIC CHANGES AFTER SPLANCHNICECTOMY

Of our patients showing preoperative fundoscopic findings of angiospastic retinitis with or without hemorrhages or exudates 82 per cent showed no evidence of angiospasm, hemorrhages or exudates at any follow-up examinations five to twelve years after operation. Papilledema usually disappeared quickly and in the twenty-one cases of malignant hypertension still living five to twelve years after operation it had not returned.

HEART CHANGES AFTER SPLANCHNICECTOMY

The response of the hypertensive heart to splanchnicectomy has been very carefully studied.[16] Three hundred eighty-four patients who had been operated upon from five to twelve years previously and who had had complete studies, including electrocardiogram, orthodiagram and teleoroentgenogram, both before and at various periods after operation, showed the following results: It was gratifying that 60 per cent of the patients with known hypertensive heart disease were still living five to twelve years after splanchnicectomy and

that 93 per cent of those who had normal hearts prior to operation were still living. Those showing abnormal preoperative electrocardiograms had significant improvement in the tracings in 41 per cent, five years or more after operation. Many patients with gross enlargement of the heart preoperatively have had return to normal size. A significant decrease in heart size has been maintained in those with definite preoperative cardiac enlargement over a five to twelve-year period in 44 per cent. Only 10 per cent of those with preoperative cardiac enlargement showed any further enlargement over the long postoperative period. Nearly all patients who have shown improvement in the electrocardiogram or decrease in heart size have maintained a significant decrease in blood pressure. As previously pointed out patients who had had congestive heart failure but who had responded to digitalis preparatory to a splanchnicectomy had a much poorer ultimate prognosis. Our recent studies have shown that only one in three of such patients will survive for a postoperative period of five to twelve years. Only 12.5 per cent of patients who had had frequent paroxysms of nocturnal dyspnea survived for this long period.

A hypertensive patient who has had a coronary occlusion with a myocardial infarction and whose blood pressure still persists at a high level presents a very serious prognosis. However, twelve such persons have been treated by splanchnicectomy without a fatality and nine of these were still living five to ten years postoperatively. This prolonged survival of 75 per cent of such severe cases is very encouraging.

Of the hypertensive patients with normal heart size prior to operation 86.6 per cent were still living and of these 92.5 per cent had maintained normal heart size.

RENAL CHANGES AFTER SPLANCHNICECTOMY

The renal functions show definite improvement in many cases following splanch-

nicectomy. Nocturia is almost completely relieved and 45 per cent of patients with abnormal urea clearance have returned to normal and 44 per cent with impaired water concentration have marked improvement. In the majority of cases albumin has completely disappeared from the urine.

PREVENTION OF CEREBRAL ACCIDENTS BY SPLANCHNICECTOMY

Excluding cases of malignant hypertension fifty-eight patients are still living five to thirteen years after operation of a total of 108 who had had definite cerebral accidents prior to operation. Fifty of the fifty-eight have had no subsequent cerebral insults throughout this long postoperative period. Ten hypertensive patients who had suffered strokes prior to operation have maintained normal blood pressure levels since splanchnicectomy, the shortest period being five years. These figures indicate that, even though the patient has had a cerebrovascular accident, surgery offers real hopes of a prolonged useful life.

PROBLEM OF SURVIVAL IN HYPERTENSION

The ultimate test for any treatment of hypertension is whether or not it alters the progressive and finally fatal course pursued by a great percentage of the cases. Our recent study of patients who had been operated upon five to thirteen years previously showed that 80 per cent of the entire series were still living at the end of five years, 57.3 per cent after ten years and 41.2 per cent after thirteen years. It must be remembered that the great majority of these patients had progressed to very serious constitutional involvement of the heart, kidneys or cerebral vessels before operation. The operative mortality of our entire series is now 1.5 per cent. Comparing the above figures with the few available statistics on the survival of hypertensive patients under the best medical management furnishes strong support for our belief that a bilateral supradiaphragmatic splanchnicectomy offers the very best form

of treatment for the patients with progressive hypertension.

MALIGNANT HYPERTENSION

The patient showing definite papilledema, usually with hemorrhages and exudates, and with a high systolic and diastolic pressure, and frequently with evidences of severe cardiac and renal damage presents an absolutely hopeless prognosis from the medical standpoint. In the Keith, Wagener, Barker[7] series of 146 patients with malignant hypertension only 21 per cent were living at the end of the first year. A similar group operated upon with a supradiaphragmatic splanchnicectomy had in contrast a survival of 64 per cent at the end of one year.[17] Those treated medically had a survival of only 12 per cent at the end of the second year while those treated surgically had a survival rate of 50 per cent after the same length of time. After four years only 2 per cent of the medically treated patients were alive contrasting with 33 per cent survival of the surgically treated patients. At the end of five years only one of 146 medically treated patients was alive, while thirty-one (21.6 per cent) of 143 cases were living five years or more after splanchnicectomy. One case with definite preoperative evidence of a severe, malignant hypertension is now living thirteen years postoperatively and the blood pressure has been maintained at a normal level.

PREGNANCY FOLLOWING SPLANCHNICECTOMY

Pregnancy presents a very serious problem in hypertension. Frequently the hypertension is present before the patient becomes pregnant but in some cases it apparently develops as a result of pregnancy. Ordinarily the hypertensive female who becomes pregnant is much less likely to go through child-bearing successfully than is the woman with a normal pressure. She seems markedly vulnerable to the toxemia of pregnancy. Also it has been estimated that about 25 per cent of women

who have a toxemia of pregnancy have a persistent postpartum hypertension.[3] We have operated upon twenty-eight hypertensive females who subsequently experienced thirty-four pregnancies.[19] Eighteen of these women had maintained normal pressures following their splanchnicectomy and before they became pregnant. Seventeen of these gave birth to eighteen living infants and fifteen of the patients were still maintaining normal blood pressure levels at intervals averaging 2.7 years following delivery and 6.3 years following splanchnicectomy. Of the 10 remaining patients whose blood pressure had not been reduced to normal only two were delivered of living infants at full term. None of the women with normal blood pressures following operation and prior to pregnancy developed a toxemia of pregnancy and none suffered any apparent damage to either the heart or the kidneys. Twelve of the women with normal pre-pregnancy blood pressures went through thirteen full term pregnancies and maintained normal blood pressure levels throughout. Mild to moderate elevations were noted during fourteen pregnancies. These included the patients who had an elevation of blood pressure at the onset of pregnancy. In five of these, living infants were delivered at full term. In only four cases (these had elevated pressures at onset of pregnancy) did the blood pressure reach or exceed the preoperative level and in each case the pregnancy was interrupted.

It is evident from the above figures that if a woman with hypertension desires to have a child she should first have a splanchnicectomy. If then her blood pressure comes down to normal and remains so for a year, we think it is safe for her to proceed with child-bearing. Her chances are excellent for delivery of a normal infant and she has little to fear from a possible toxemia of pregnancy or from any late postpartum sequelae associated with her previous hypertension. We have also performed a splanchnicectomy upon five women during the second trimester of their pregnancies because of exceedingly high blood pressures

and symptoms of the toxemia of pregnancy.[18] It is generally advised to interrupt such a pregnancy. However, this may not be necessary if a splanchnicectomy is performed. In two of the five cases brilliant results were obtained. In both the blood pressure returned to normal, the toxemia disappeared and normal living infants were delivered at full term. In these two women the normal blood pressure levels have been maintained for four years and two years, respectively, since the delivery. In the other three cases the splanchnicectomy apparently had no influence on the toxemia but in one of these cases the blood pressure levels since delivery have been significantly reduced.

RELIEF OF INCAPACITATION

Incapacitation, either because of the severe symptoms of hypertension or because of organic changes in the heart or the kidneys, has been significantly relieved in a large number of our patients by splanchnicectomy.[14] The ability of these patients to resume gainful work following operation is one of its most gratifying results. In a follow-up study covering seven years, 55.5 per cent of the patients who had been incapacitated had had complete recovery from such incapacitation and had returned to their former occupations. Many others had shown so much improvement that they were again employed although not at their original jobs. This makes a total improvement of 81.3 per cent. As a rule the patients are able to return to work within a few weeks following operation.

CASE REPORTS

The following three case histories are given to illustrate typical results obtained by bilateral supradiaphragmatic splanchnicectomy in patients with malignant hypertension, severe angina and pregnancy following splanchnicectomy.

CASE. 1. *Malignant hypertension:* V. E., age twenty-two, was known to have an elevated blood pressure for three years. On entrance she complained of severe headaches, swollen

ankles and blurred vision. The patient had developed severe precordial pain in the left arm, nausea and vomiting, and had been confined to bed for eight months because of the severity of her symptoms. In spite of this long rest her blood pressure averaged 280 systolic, systolic, 90 diastolic. She was still entirely free of symptoms. Electrocardiogram and heart size were normal. At no time had there been any evidence of retinal disturbance. Twelve years postoperatively her blood pressure was 128 systolic, 90 diastolic. She was still completely

SYMPTOMS   ONSET OF HYPERTENSION 3 YEARS PREVIOUSLY WITH TOXEMIA OF PREGNANCY. SEVERE HEADACHES. NAUSEA AND VOMITING. BLURRED VISION. COMPLETE INCAPACITATION. CONFINED TO BED.

SYSTOLIC BLOOD PRESSURE: 280, 110, 135, 144, 140, 140, 140, 130, 130, 128, 110

DIASTOLIC BLOOD PRESSURE: 190, 90, 90, 110, 106, 96, 90, 90, 90, 77

MAXIMUM CONCENTRATION: 1.033, 1.029, 1.027, 1.026, 1.026, 1.023, 1.019, 1.016, 1.016, 1.009

UREA CLEARANCE %: 87, 85, 73, 77, 75, 94, 86, 105, 94

URINE PROTEIN %: 0.12, 0.02

URINE RBC

FUNDI: PAPILLEDEMA, HEMORRHAGES, EXUDATE, ANGIOSPASMS

TIME IN YEARS: OPR. 1 2 3 4 5 6 7 8 9 10 11 12 13

FIG. 1. Chart illustrating results of bilateral supradiaphragmatic splanchnicectomy and lower dorsal sympathetic ganglionectomy during a thirteen-year postoperative period. (Case 1.)

190 diastolic in each arm. Funduscopic examination showed a severe neuroretinitis with early papilledema, flame-shaped hemorrhages and cotton wool patches. There was localized angiospasm and arteriovenous compression. The electrocardiogram showed inverted T waves in leads II and III. She had a proteinuria of 0.12 per cent and a moderate number of red blood cells and casts in the urine. Water concentration and urea clearance were normal. On June 22, 1934, a bilateral supradiaphragmatic splanchnicectomy was performed. She was discharged on the fourteenth postoperative day.

An examination two months later showed that the retinal hemorrhages, exudates and papilledema had completely disappeared. The blood pressure was 145 systolic, 90 diastolic. One year postoperatively her blood pressure was 135 systolic, 90 diastolic. She was completely asymptomatic. Ocular fundi examination was entirely normal. She was examined at yearly intervals with similar findings. At the end of ten years her blood pressure was 130

asymptomatic. Electrocardiogram and heart size were normal. Thirteen years following operation the blood pressure was still normal at 110 systolic, 77 diastolic. (Fig. 1.) Funduscopic examination, electrocardiogram and orthodiagram were all normal. She was entirely symptom-free.

CASE II. *Hypertension with severe angina pectoris:* M. S., age forty, entered the hospital on September 2, 1941, complaining of episodes of excrutiating pain across the upper chest always related to exertion and disappearing five to fifteen minutes after rest. She had been a known hypertensive for ten years. For the past month she had had from two to six episodes of very severe angina pectoris daily. Her blood pressure averaged 230 systolic, 150 diastolic. Funduscopic examination showed moderate arteriosclerosis and localized angiospasms. There were no hemorrhages or exudates. Electrocardiogram showed deeply inverted T waves in leads I and II and in chest leads evidence of anterior myocardial infarction. Heart size was normal. She

was continued at bed rest and averaged two anginal seizures daily. On September 18, 1941, she had a protracted episode of very severe substernal pain requiring morphine and the following day her electrocardiogram showed prominent T waves in leads II and III and evidences of a new posterior myocardial infarction. The blood pressure remained high. On October 17, 1941, in spite of the fact that she had had two comparatively recent myocardial infarcts a bilateral supradiaphragmatic splanchnicectomy was performed and she had an uneventful postoperative course. Five years postoperatively her blood pressure was 164 systolic, 114 diastolic. Electrocardiogram showed prominent Q waves in leads II and III. Heart size was normal. She has not had a single anginal attack since operation.

CASE III. *Pregnancy after a splanchnicectomy:* J. T., age twenty-three, had a known hypertension of two years. Blood pressure averaged 230 systolic, 145 diastolic. Funduscopic examination showed occasional localized angiospasm and marked attenuation of the retinal arterioles. One cotton wool exudate was noted. Electrocardiogram and heart size were normal. The kidney function tests were normal. On April 20, 1938, a bilateral supradiaphragmatic splanchnicectomy was performed. Two years postoperatively her blood pressure was 126 systolic, 82 diastolic. There had been complete relief of the very severe preoperative headaches. Five years after splanchnicectomy she completed a full term pregnancy with a normal living infant. Her blood pressure had remained normal throughout the pregnancy. There had been no albumin in the urine. Seven years after operation her blood pressure was 118 systolic, 86 diastolic. She was completely free of symptoms; the electrocardiogram and heart size were normal. Kidney function was also normal.

SUMMARY

The symptomatology of hypertension is discussed at length and its possible bearing on prognosis given. The etiology of hypertension is considered, especially in reference to its surgical treatment.

A bilateral supradiaphragmatic splanchnicectomy is recommended as the procedure of choice in patients showing a progressive type of hypertension or in those who have already reached a high level. The results of careful studies over a long period show significant improvement as measured by a reduction in the blood pressure, improvement of ocular, renal and cardiac status, alleviation of symptoms, relief of incapacitation and a probable prolongation of life.

Approximately 2,000 patients have been operated upon by this one procedure at the University Hospital during the past fourteen years and a sufficient number of these have been studied over a long enough period to make definite statements possible.

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TUMORS OF THE SPINAL CORD

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IN recent years, with the discovery that many of the heretofore unexplained pains in the upper and lower extremities were in reality due to protruded intervertebral disks, other intraspinal lesions have been overshadowed and so may be overlooked in the making of a differential diagnosis. The reason for such a statement is that neurologists and neurosurgeons are seeing an increasing number of patients who have been treated for protruded intervertebral disks, only to find that the symptoms have persisted rather than abated. Much to the chagrin of everybody concerned, tumors of the spinal cord subsequently have been removed at operation in such instances, and the symptoms have been relieved thereby.

Intraspinal lesions which produce symptoms of weakness, numbness and pain involving the neck and shoulders, thorax, abdomen, pelvis and extremities create a fascinating and stimulating clinical problem. These lesions may vary from inflammatory adhesions and scars to vascular anomalies and varicosities. They may consist of tumors arising from the bone, fat, cartilage, fibrous tissue and nervous tissue of the extradural space. These tumors may arise also from the arachnoid, blood vessels and nerves of the intradural, extramedullary region, and they may arise from the spinal cord itself, originating either in the fibrous or the neurogenic tissue.

The surgical approach to intraspinal lesions dates back to the time of Hippocrates, but the classical description of the development of symptoms is contained in the report of the first successful removal of a tumor of the spinal cord. Dr. William Gowers and Sir Victor Horsley published this report in the *Medico-Chirurgical Transactions* in 1888, and it is so succinct, so comprehensive and so stimulating that even today it remains a classic.

Since Horsley's epoch-making operation, the diagnosis of tumor of the spinal cord has been made in many cases and the tumors have been successfully removed. Even with the experience gained in hundreds of these cases, however, there still remains an interval between the onset of pain and the development of other symptoms, during which time many diagnoses may be hazarded, and different varieties of treatment may be instituted.

The medical profession at large has learned to accept the fact that when the diagnosis is made early and the tumors are completely removed, excellent clinical results may be obtained. But early diagnosis often is difficult, especially when the only symptom is intermittent pain, sometimes projected to some definite, localized region in the thorax, abdomen or extremities. When the pain is associated with paresis or anesthesia, an organic neurologic lesion is suggested immediately. However, patients sometimes require relief of pain before any associated motor or sensory defect can be determined, and that pain may so simulate the pain of other lesions in the body that radical therapeutic measures are instituted which prove to be ineffectual. Consequently, patients suffering from early pain caused by intraspinal tumors have had gallbladders, appendixes, fallopian tubes and uteri removed, yet, even when a definite pathologic condition could be demonstrated, the pain has recurred. The probability that such operations would be carried out at first seemed rather remote; but in a review of a large series of cases of

proved tumors of the spinal cord, it was found that such measures to relieve the early symptoms of pain actually had been taken in about 10 per cent of the cases, and that the patients concerned ultimately had undergone removal of tumors.

Instances in which such operations were carried out not only represented attempts to relieve pain which, originating from lesions in the spinal cord, was projected to certain other regions, but also demonstrated the possibility of coexisting pathologic change which might be symptomless. Thus, for example, cystic ovaries may be discovered in cases in which pain is projected to the pelvis, and likewise, fibromyomas of the uterus can be demonstrated in cases of painful back associated with pain projected to the pelvis. Evidences of inflammatory processes about the appendix and gallbladder can be demonstrated in cases in which pain is projected to the right lower and right upper abdominal quadrants. But the unsatisfactory attempts to relieve the pain should suggest that the underlying pathologic process involved the spinal cord.

SYMPTOMS

The symptoms of intraspinal lesions have been divided by Oppenheim and by Frazier into three stages, which the authors designated as "cycles." The *first cycle* is the root, or pain cycle, which usually is the longest in the period of the disease. It is not uncommon for patients suffering from root pain to present themselves to their family physician for examination complaining of rheumatism, neuritis and miscellaneous abdominal disturbances. Physicians and surgeons may be tempted to treat these complaints without making a thorough neurologic examination.

Woltman has stressed the point that almost no pain is pathognomonic, but that a given disease may be accompanied by many different kinds of pain; and that although pain may be the only symptom, yet much may be learned by inquiry as to the mode of onset, the duration and the intensity. Pain is a subjective complaint. Pain is something that cannot be seen or palpated, which means that cautious interpretation of what the patient has to tell is necessary. Seven points at least should be determined regarding each complaint of pain: (1) situation, depth and projection, taken together, (2) frequency, (3) duration, (4) character, (5) intensity, (6) progress and (7) associated symptoms.

In the making of a differential diagnosis between pain caused by intraspinal lesions and pain caused by organic lesions of the thorax, abdomen and extremities, many procedures of diagnostic value are available. None of these can begin to compare with a careful and thorough neurologic examination. For such an examination it is necessary to have the patient disrobe completely. The physician must consider any change from normal in the many reflexes, in the response to cutaneous stimulation by touch, heat and cold, and in the strength of the muscles. Roentgenologic examination of the spinal column frequently is of great value in demonstration of the presence or absence of changes in the bony structure caused by inflammation, previous trauma, erosions and tumors. Examination of a specimen of cerebrospinal fluid obtained by lumbar puncture is one of the most valuable diagnostic procedures. The physical, chemical and cytologic characteristics of the fluid may contribute the evidence necessary for diagnosis of an intraspinal lesion. Additional examination of changes in pressure, after an increase in intracranial pressure resulting from compression of both jugular veins, determines the presence of subarachnoid block, which prevents free circulation of the fluid in the subarachnoid space. This sign, coupled with the pathognomonic pain of tumor of the spinal cord, frequently is the means of arrival at an early diagnosis in cases in which an intraspinal lesion is suspected.

In the presence of pain and positive results of examination of the cerebrospinal fluid, it is often very difficult to localize the lesion of the spinal cord sufficiently ac-

curately to enable relief to be given by surgical means. As a means of further differential diagnosis, iodized oil can be used. When this opaque substance is injected within the subarachnoid space, it collects at the level of the block, and this level can be visualized by means of roentgen rays. It is often important to make a very careful neurologic examination after the removal of cerebrospinal fluid, inasmuch as levels at which sensory disturbances appear may become apparent, reflex changes may take place and muscular weakness may occur. With all these diagnostic procedures at the physician's command, it still may be impossible to demonstrate any signs of a neurologic lesion. In such an instance, examination should be repeated at frequent intervals before any drastic therapeutic measures are indicated for the relief of pain in patients suspected of having intraspinal lesions.

We often refer to organic lesions as producing characteristic symptoms which should arouse a suspicion of the underlying lesion. For instance, we say that tumors of the brain produce the three cardinal signs of headache, nausea and vomiting, and increased intracranial pressure. So, also, we speak of intraspinal lesions as giving rise to characteristic symptoms. Pain is the outstanding symptom of intraspinal involvement of nerve roots, and may be characteristic, sometimes being preceded by symptoms of irritation such as paresthesia, hyperesthesia or complete anesthesia in the area supplied by the root or roots. The pain of an intraspinal lesion may precede any other symptom by months or years; it may be constant or intermittent; its chief characteristic is that it occurs when the patient is at rest and is relieved by exercise. The character of the pain is almost pathognomonic since it persists in a localized area and extends over the same nerve roots. It is usually lancinating and is aggravated by coughing, sneezing, lifting and straining at stool. It invariably awakens the patient from four to six hours after retiring. It often becomes so severe

as to compel the patient to walk the floor or to sleep in a sitting position. The mechanism of the production of this pain apparently is brought about by the ball-valve action of the tumor, which is forced downward by the increased pressure of the cerebrospinal fluid above the tumor, thus producing traction directly or indirectly upon the nerve roots. Unfortunately, many of the patients are treated for neuritis, muscular rheumatism or syphilis, and some have even been called "hysterical." The importance of the recognition or suspicion of the first or painful phase in the development of spinal cord tumors was emphasized in a recent survey in which 10 per cent of the patients presenting root pain had been operated upon for some thoracic or abdominal lesion other than that of intraspinal tumor.

In the discussion of tumors of the spinal cord it is usual to consider the tumors as they occur anatomically. When the tumor occurs in the *cervical region* of the spinal cord, the pain may be in the neck or it may extend to the shoulders. The pain may be increased by laughing, sneezing and coughing, and may be accompanied by some rigidity of the neck. Tumors in the upper part of the cervical region may produce symptoms similar to those of involvement of the medulla oblongata. Paralysis of the diaphragm may occur when the fourth cervical segment is involved. Tumors of the lower part of the cervical enlargement may cause atrophic paralysis of the small muscles of the hands. The oculopupillary symptoms (contraction of the pupil, narrowing of the palpebral fissure and enophthalmos) are present on the side of the lesion.

Tumors which are compressing the *thoracic segment of the spinal cord* usually cause pain which frequently simulates that of intercostal neuralgia, cholecystitis or cholelithiasis, renal colic or appendicitis. The zone of hyperesthesia is above the level of the lesion.

Tumors situated in the *lumbar and sacral portions of the spinal cord* present

difficulties in differential diagnosis because a neoplasm in this part of the vertebral canal can compress the fibers of the cauda equina and produce practically the same symptoms. Symptoms generally are those produced by compression of the spinal cord. Pain is outstanding, frequently is diagnosed as "sciatica," and may be present for months and years before there are any localizing signs. Functions of the bladder and rectum are interfered with early in tumors of the conus medullaris. These tumors do not cause paralysis unless they grow to such an extent as to compress the roots of the cauda equina. Disease of the conus medullaris is characterized by rapid development, absence of pain, appearance of anesthesia of an associated character, motor irritative phenomena such as fibrillary twitchings, and general and rapid development of paralysis of the bladder, rectum and intestinal musculature.

For convenience in discussing the symptomatologic aspects of intraspinal lesions, a *second cycle* or phase has been described. The symptoms which develop in this second phase differ from those of the first in that neurologic evidence of compression of the spinal cord now becomes evident. These symptoms may develop simultaneously with the existence of pain or they may develop without the presence of pain. If the tumor is situated anterolaterally, the symptoms will progress and produce the classic Brown-Séquard syndrome. This is the characteristic symptomatology of the second phase. It consists of homolateral paralysis or paresis of the muscles below the level of the lesion, with a loss of, or diminution in, sensation of pain and temperature on the opposite side. This syndrome is common in cases of extramedullary tumors because most of these tumors exert their pressure on the anterolateral and posterolateral aspects of the cord. If the posterior columns of the spinal cord are interfered with, deep sensibility is decreased, and consequently ataxia in the extremities and below the lesion occurs. Sensory disturbances caused by compression of the spinal cord develop in an ascending manner; after removal of the lesion normal perceptive powers are restored in a descending sequence.

Tumors of the upper and lower ends of the spinal cord may present certain peculiar features. The crossing of the pyramidal tracts at the anterior aspect of the cord in an exposed situation may result in quadriplegia unaccompanied by any sensory disturbance. The sensory disturbance associated with high cervical tumors may be limited to the lower extremities; it may fluctuate from time to time and from place to place, probably because the relatively large cervical canal permits shifting of the pressure. The proximity of the foramen occipitale magnum may bring about signs of a tumor of the brain, such as a projectile type of vomiting and choked optic disks.

When a tumor occurs at the lower end of the spinal cord, other difficulties may be encountered. The relative shortening of the cord incident to growth and the emergence of the roots through the anterior foramina of the sacrum often make it extremely difficult to determine whether there is a tumor of the cord or the cauda equina, or a tumor situated in the pelvis. The objective findings may be the same.

Again, the *third cycle* or phase in the symptomatologic aspects of intraspinal tumors may be characteristic of the progress of the disease. Paralysis occurring below the level of the tumor comprises this third phase. It is caused by extreme compression of the cord. The paralysis usually is complete, for both motor and sensory functions and trophic disturbances are present, as well as definite loss of both vesical and rectal sphincter control.

EXAMINATION

After the taking and recording of a history, a detailed general, as well as a neurologic, examination is necessary. This should include such special examinations as spinal puncture and the making of roent-

genograms of the spinal column, with or without the introduction of iodized oil.

General Examination. In the presence of all suspected intraspinal lesions, close inspection of the skin of the body is necessary for evidences of cutaneous fibromas or the pigmented patches of multiple neurofibromas (von Recklinghausen's disease). Primary malignant lesions of the abdominal and pelvic viscera should be kept in mind when patients are elderly, and in such instances the urine should be examined for evidence of renal tumors. Dumb-bell tumors have been found with roentgenologic evidence of tumors within the thorax indicative of this type of compression of the spinal cord.

Roentgenologic Examination. Roentgenograms should be made of the anterior, posterior and lateral aspects of the vertebral column. These roentgenograms should be supplemented by stereoscopic and oblique views made at the level suspected clinically. According to Camp and Adson evidence of erosion of the vertebral pedicles, laminas and lateral and spinous processes resulting from pressure usually is discernible before comparable evidence is apparent in the bodies of the vertebrae. In general, the roentgenologic evidence of changes caused by tumors of the spinal cord consists of erosion secondary to direct pressure, invasion by the tumor, destruction brought about by benign or malignant tumor of the bone, metastatic disease and hyperostosis.

Camp, in a review of cases, reported that osseous changes which directly localized the lesion were observed roentgenographically in 30.3 per cent of the cases. He divided tumors of the spinal cord into three groups roentgenologically.

The first, and fortunately the most predominant group, are benign tumors arising from the soft tissues within the spinal cord. The most common in this group include neurofibroma, endothelioma, hemangioma, angioma, lipoma and dermoid cyst.

The second group of tumors classified roentgenologically consist of malignant tumors arising from the soft tissues within the spinal canal. These usually are metastatic and as a rule are diagnosed by means of roentgenograms.

The third group comprise the benign tumors of the vertebrae, including osteomas, osteochondromas, chondromas, fibrochondromas, giant-cell tumors and hemangiomas.

Roentgenologic diagnosis can be made in the presence of primary infections of the vertebra, such as tuberculosis, osteomyelitis, actinomycosis and coccidioidal granuloma, which produce destructive areas in the vertebra, together with involvement of intervertebral disks.

Of distinct value is roentgenologic examination of the intervertebral canal with contrast media. In addition to the roentgenologic evidence of tumors, which is apparent in routine examination of the cord, roentgenoscopic and roentgenographic study by the use of radiopaque oil furnishes considerable information. Injection of 5 cc, of iodized oil into the subarachnoid space. either through cisternal puncture or by means of lumbar puncture, allows visualization of the patency of the subarachnoid space under the roentgenoscope. Roentgenoscopic examination of slowly moving oil is superior to study of the roentgenogram, since the physician often is able to see the diversion of the oil current around the tumor, although roentgenograms should be made for confirmation of the level of the lesion. Intramedullary tumors are identified by a division of the oil into two currents. After the oil has been injected, the patient should be placed prone on the roentgenoscopic table and the flow of oil should be observed with the patient in the various positions, from the horizontal to the perpendicular. Experience with the use of radiopaque oil in the diagnosis of tumor of the spinal cord has indicated that it should be used only in conjunction with a complete examination, and that the oil should be removed immediately after the need for it has passed. The iodized oils most frequently used are panto-

paque and lipiodol. Pantopaque is employed more often in the differential diagnosis when protruded intervertebral disks are suspected. Camp and his colleagues have called attention to the fact that when a tumor is suspected to be situated above the conus medullaris, the use of lipiodol allows for a more nearly exact examination.

Neurologic Examination. When intraspinal tumor is suspected, there is no examination so important as a complete neurologic examination. The information elicited by a detailed testing of reflexes, muscle strength and tonus, sensory acuity, as well as gait, co-ordination and balance, tends to distinguish between degenerative diseases and the compression syndrome of intraspinal tumor.

Since the spinal nerves carry both sensory and motor impulses, a lesion of these will manifest itself by disturbance of all forms of sensation and motility. Involvement of a posterior root alone will result in disturbance of all forms of sensation; involvement of an anterior root alone will produce only a motor disturbance. After entry of the posterior root into the cord, however, the various forms of sensation pursue different paths, so that a lesion situated within the spinal cord might not affect all forms of sensation.

Fibers which carry sensations of pain and temperature are closely associated. They may be regarded as taking the same course. They cross almost immediately to the opposite side of the cord in the anterior white commissure, and ascend to the brain in the lateral spinothalamic tract.

Tactile stimuli take two pathways: one proceeds up the posterior column on the same side of the cord, but crosses to the opposite side after it reaches the brain stem; the other crosses almost immediately and ascends in the anterior spinothalamic tract of the opposite side. Therefore, a lesion of either half of the cord alone will not abolish tactile sensation completely.

Motor impulses arise in the brain, leave the pyramidal cells of the prerolandic area, and pass downward in the pyramidal tract, which crosses to the opposite side about 1 cm. below the pons cerebelli and descends in the lateral column of the cord. A lesion of the pyramidal tract results in the form of paralysis seen in ordinary hemiplegia. The muscles are not wasted; they are hypertonic, and the tendon reflexes are more active than is normal. The plantar response elicited by the Babinski method usually is extensor; that is, when the sole of the foot is stroked, the great toe turns up.

The motor impulses leave the pyramidal tract and are relayed to the cells of the anterior horn, or lower motor neuron, on the same side. A lesion of the lower motor neuron results in a form of paralysis differing in many respects from that caused by involvement of the upper motor neuron and resembling that seen in poliomyelitis. The muscle fibers supplied by the corresponding neurons are weak and atrophied, and the tendon reflexes are reduced to a point commensurate with the amount of muscle substance remaining.

Since, in compression of the spinal cord, one side usually is affected a little before the other, the so-called Brown-Séquard syndrome is produced. The cardinal features of this syndrome are: (1) homolateral paralysis below the given level, caused by involvement of the pyramidal tract, (2) impairment of pain and sensation of temperature of the opposite side below the level in question and (3) preservation of tactile sensibility on both sides.

The segmental distribution of cutaneous sensation is very orderly. The various fields of sensation supplied by the different segments of the spinal cord can be mapped out and shown to be running in long bands down the arms, in circles about the trunk, in bands down the legs, and ending in the concentric rings around the anus. Actually, these segments overlap each other like shingles on a house, so that two or three adjacent roots would have to be destroyed before the area of anesthesia could be demonstrated.

Much interest has been evidenced recently in the determination of the upper levels of lesions and tumors of the spinal cord by means of vasomotor lines of demarcation. Vasomotor lines of demarcation frequently will indicate the level of the lesion before the development of sensory or motor symptoms of focal value. In many instances pilocarpine has been used to stimulate sweating; and the reaction indicates that in cases of intramedullary disease, the sweating response rarely indicates a level of any diagnostic value. In cases of extramedullary disease (tumor), the sweating reaction tends to divide the body into contrasting areas, meaning that there is very little sweating below the level of the lesion. As a rule, in extramedullary disease the segmental level below which sweating is suppressed corresponds with the level below which sensation is diminished. The segmental representation of the reflexes also aids in determination of the site of the involved portion of the spinal cord.

Spinal Puncture. Examination of the cerebrospinal fluid discloses information concerning the physical and hydrodynamic properties and the chemical reactions of the cerebrospinal fluid. Spinal puncture generally is performed at the fourth lumbar interspace. Before any fluid is removed, the intraspinal pressure is estimated by means of a manometer which normally registers between 12 and 15 cm. of water. As soon as the pressure has been estimated, Queckenstedt's test is made. This consists of reading and study of the rate of increase of the cerebrospinal fluid in the manometer after compression of both internal jugular veins. The sudden increase and rapid decrease of the fluid on compression of both internal jugular veins indicates a free flow of cerebrospinal fluid within the subarachnoid space, and rules out the possibility of a compressive mechanism. A slow increase and decrease of fluid or failure of the fluid to rise at all on compression on the jugular veins suggests partial or complete intraspinal block.

Inability to obtain fluid at the fourth lumbar interspace may signify that the subarachnoid space has not been entered, or absence of fluid or the presence of a tumor at this level. In such a circumstance other levels should be used, and it may be necessary to make multiple punctures which may localize the tumor definitely.

Examination of the cerebrospinal fluid with regard to the number and kind of cells and the amount of protein, and also other chemical tests, as well as the colloidal gold curve, may be methods of distinguishing between intraspinal lesions and degenerative lesions, such as multiple sclerosis.

It must be remembered that the presence of partial or total subarachnoid block is not pathognomonic of intraspinal tumor since previous attacks of meningitis, acute myelitis, injuries to the vertebrae or spinal deformities are all capable of interfering with the free flow of cerebrospinal fluid. But it is apparent that subarachnoid block is extremely valuable diagnostically when it is found in conjunction with a history of root pain without inflammation or trauma to the spinal cord.

Differential Diagnosis. The physician must always keep in mind, when a tumor of the spinal cord is suspected, the possibility of intraspinal metastatic lesions. For that reason, a complete general examination is important. Carcinomas of the breast and prostate gland head the list of metastatic lesions to the spinal cord. It must not be forgotten that a primary carcinoma of the breast may have been removed many years before metastasis to the cord gives evidence of its presence. Since carcinoma of the prostate gland may not produce local symptoms, rectal examination should be made.

Meningomyelitis is another condition which may remain localized, and in which there is often a history of antecedent trauma or infection.

The spinal column itself may be the seat of the original disturbance which causes pressure on the spinal cord. This may occur as the result of *Pott's disease, chronic hyper-*

trophic osteo-arthritis, spondylolisthesis, or a slipping forward of a vertebra—usually the fifth lumbar vertebra—on the sacrum, and *Paget's disease*.

Spina bifida may be associated with a superimposed tumor.

In cases of *subacute combined degeneration* of the spinal cord, the lesion, although progressive, as a rule is painless and diffuse.

The diagnosis of *tabes dorsalis* sometimes is made when there are tumors of the spinal cord or cauda equina, probably because the patellar reflexes may be absent, but a complete examination usually indicates the true nature of the disease.

It may be difficult to distinguish *syringomyelia* from an intramedullary tumor of the spinal cord. Aids to correct diagnosis are the characteristic waistcoat type of sensory disturbance, which seems to affect chiefly pain and temperature fibers, since these lie near the central canal; the local atrophy caused by extension of the process here and there into the anterior horns, and the tell-tale scoliosis.

Although *multiple sclerosis* may produce a transverse lesion of the spinal cord, the youth of the patient, the absence of pain and the presence of cerebral signs, such as tremor, scanning speech, nystagmus, optic atrophy, ocular palsy and the characteristic emotional lability generally put the physician on guard.

SURGICAL TREATMENT

Anesthesia. The selection of the proper anesthetic agent to use during operations on the spinal cord depends on the patient and the facilities for administration. Most patients prefer general anesthesia. Ether dropped onto an open mask or over a Magill intratracheal tube is to be preferred to ethylene or nitrous oxide. Paravertebral regional anesthesia produced with procaine hydrochloride and epinephrine minimizes the amount of bleeding, but cannot be used for hypersensitive patients. Tribromethyl alcohol (avertin) has been used successfully, although occasionally an idiosyncrasy toward the drug has proved alarming.

Operation. Once the diagnosis of compression of the spinal cord by a tumor of the cord has been established, the treatment is essentially surgical, because the object is relief of compression of the spinal cord. The surgical mortality rate associated with removal of tumors of the spinal cord is less than 4 per cent. Exposure of the spinal cord at operation is accompanied by so little risk that it can be carried out routinely in the presence of dysfunction of the spinal cord associated with a distinct sensory level and subarachnoid block, or when the level has been established by means of iodized oil. Good exposure of the cord is an essential factor, and, of course, the size and extent of the tumor necessarily control the extent of the laminectomy. The mechanical removal of the spinous processes and laminas is not so important as is close observation of the tissue removed. The texture of the removed bone should be scrutinized carefully for evidences of any changes, either of absorption or of overgrowth. Then the epidural space requires critical survey for signs of neoplastic or inflammatory change. If the results of exploration have been negative up to this point, the dura should be carefully examined for any abnormalities of pulsation. Absence of pulsation means that one of two conditions is present: either the compression is situated above the laminectomy opening or the exposed dura lies just over the tumor. Gentle palpation of the dura rules out the latter condition; and if there are no signs of compression, the laminectomy must be carried cephalad until there is a definite pulsation of the dura. During the operation, it must be kept in mind that accurate as the localization has been, there is always danger that the level of the abnormality, as determined before operation, may be one segment lower than the actual compression of the cord.

Extramedullary tumors are apparent by this time in the carrying out of the operation, and should no evidence of compression be found to be caused by extradural

lesions, the dura can be opened and the cord examined. A silver probe or soft rubber catheter can be gently inserted intradurally to eliminate the possibility of an obstruction, either above or below.

Intramedullary lesions, or those situated within the spinal cord, may cause symptoms of compression. The majority of these lesions involve a discouraging prognosis, yet cysts have been evacuated, benign tumors have been completely removed and recovery has resulted from the decompression incidental to the laminectomy.

There are certain non-neoplastic lesions which have to be kept in mind in the performance of laminectomy for compression. Pachymeningitis brought about by tuberculosis, syphilis or other chronic inflammatory lesions may prove to be the cause of the compression. Chronic cystic arachnoiditis may be encountered when the dura is opened. Varicosities of the meningeal vessels may simulate tumors, although such a condition may be found adjacent to tumors, making the diagnosis of varicosity possible only after the presence of a tumor definitely has been excluded. The surgical and pathologic aspects of compression of the spinal cord are so closely interwoven that until a complete examination of the vertebrae, meninges and cord has been carried out, the operation should not be terminated or the diagnosis completed.

A linear incision is made in the skin immediately over the spinous processes which are to be removed. The incision should extend at least one segment or two segments above and below. Subperiosteal dissection of the periosteum of the spinous processes and the laminas in conjunction with the erector spinae muscles controls the bleeding and permits more efficacious closure than would incision of the muscle attachments. After the muscles have been dissected from the laminas and are held in place by self-retaining retractors, the bone is carefully removed, resection being carried out fairly wide on each side to prevent subsequent compression on the cord by calluses which may develop. Prominent spinous processes above and below the site of laminectomy should be resected in order to avoid any undue prominences in the wound. In the removal of laminas over a tumor, extreme care must be used to avoid additional pressure on the cord and accidental trauma or interference with the circulation by sponging and dissection. If the tumor is situated anterolaterally or anteriorly, it is better to section it and remove it piecemeal rather than to attempt to force it out intact at the risk of injury to the cord. If, after careful inspection of the meninges, there is no evidence of an extramedullary tumor and the enlargement of the cord indicates a tumor arising within the cord, a longitudinal incision to one side of the midline can be made; if the tumor is cystic, the contents of the cavity can be aspirated and sometimes the wall of the cyst can be removed. Solid tumors sometimes can be totally or partially removed, but additional trauma is to be guarded against. In closure of the wound, the dura should be sutured, unless by so doing pressure would be exerted on the cord. The bases of tumors attached to the dura should, of course, be resected. This will leave a defect in the dura. When it is impossible to close the dura, animal membrane or polythene should be used to cover the defect, so that the entrance of blood into the subarachnoid or dural spaces can be prevented. The muscles should be approximated by interrupted sutures of catgut, as should also the fascial planes and skin. Drainage of laminectomy wounds should be done at the discretion of the surgeon; and although it has been considered dangerous from the standpoint of secondary infection, the use of Penrose drainage for twenty-four hours sometimes prevents postoperative hemorrhage which would necessitate a secondary opening.

The fundamental principles underlying laminectomy are the same whether the operation is performed in the cervical, thoracic or lumbar regions. However, wherever possible, unilateral laminectomy should be done in the cervical region to

prevent the slipping of one vertebra upon another as a postoperative complication.

POSTOPERATIVE CARE

After the operation the patient is placed in bed in the horizontal position. Pillows are used to avoid undue pressure on the shoulders and hips. The patient should be turned from side to side and on the abdomen and not on the back, for the latter action would produce pressure on the wound. One of the frequent postoperative complications is inability to void. If, after twenty-four hours, the patient cannot void, one ampule (1 cc.) of prostigmine methylsulfate (1:2,000 solution) should be administered hypodermically, and if this does not cause voiding, the injection may be repeated in four hours. If this fails, one ampule (1 cc.) of doryl (acetylcholine chloride) may be given a trial. However, patients frequently complain of severe abdominal cramps after the use of this agent. If, in spite of the aforementioned measures, there is still no tendency toward voluntary voiding, the patient should be catheterized. If, then, the bladder still does not empty after catheterization has been carried out once or twice, the use of an indwelling catheter for a week or ten days is safer than daily catheterization. This indwelling catheter should be replaced every four or five days, and the bladder should be irrigated twice daily with an antiseptic solution, usually boric acid solution. Each time the indwelling catheter is replaced, the introduction of a few cubic centimeters of a 2 per cent solution of merbromin (mercurochrome) into the bladder before removal of the catheter sometimes will cause a return of vesical function if the patient is allowed to return the drug before another catheter is inserted. Doses of mineral oil or milk of magnesia are administered daily to prevent distention of the bowel or fecal impaction. In addition to this, a daily enema is sometimes necessary. In order to assure satisfactory healing, the patient should be kept in bed for ten days to two weeks. He should be permitted to sit up-right in bed for a day or two before he is allowed to be up in a wheelchair or attempting to walk.

The recovery of motor, sensory, vesical, rectal and sexual functions takes place in the reverse order of their development. The time for recovery of loss of motor and sensory functions depends on the character of the tumor and the degree and duration of paralysis. Hard, rounded tumors cause greater injury to the spinal cord and more paralysis than do the soft, elongated tumors. A 25 per cent loss of function usually is recovered within three months; a 50 per cent loss requires from six to twelve months; a 75 per cent loss requires up to eighteen months; and a total loss requires up to two years unless the injury to the cord has been so extensive that recovery will never take place. The removal of intramedullary, infiltrating tumors often results in temporary improvement which may continue for six to seven years.

PATHOLOGIC ASPECTS OF TUMORS OF THE SPINAL CORD

We have already referred to the classical descriptions of the first instance of removal of a tumor of the spinal cord. The lesion in question proved to be an extradural fibroma; consequently the return of the patient's condition to normal was rather speedy. The anatomic situation of the tumor in respect to the meninges is important in the making of the prognosis. However, pathologically, it is important to note the anatomic situation of the tumor with regard to segments of the spinal cord. It is interesting to observe that, anatomically, the majority of tumors of the spinal cord occur in the thoracic region. This might be explained by the fact that the thoracic segments comprise more than half of the entire length of the cord.

Five hundred fifty-seven verified intraspinal neoplasms were classified pathologically and grouped according to situation by Rasmussen, Kernohan and Adson. Neurofibromas constituted the largest single group. Meningiomas, or arachnoid fibro-

blastomas, comprised the second largest group, and the primary distribution of these was found to be in the thoracic region. Ependymomas were noted to be fairly evenly distributed throughout the spinal cord. Half of them, it appeared, arise from the spinal cord proper, and the remaining half arise from the filum terminale. Vascular tumors form a group which includes the hemangio-endotheliomas and hemangiomas. Chordomas were found to occur most often in the sacral region. Sarcomas, under the heading "sarcomas," were included by Rasmussen, Kernohan and Adson in a miscellaneous group of fifty-five sarcomatous lesions consisting of lymphosarcomas, myelosarcomas, giant-cell sarcomas, Hodgkin's disease and osteogenic sarcoma.

EXTRADURAL LESIONS

The extradural lesions (Fig. 1) include inflammatory lesions, tuberculosis and tuberculomas, hypertrophic osteitis and metastatic tumors, such as carcinomas, fibromyxosarcomas, fibrosarcomas and hypernephromas.

Extradural gliomas are very rare; they probably are extensions, into the epidural space, of tumors of the spinal cord.

Hemangiomas and hemangio-endotheliomas may occur extradurally; they arise from the vessels which normally are found lying extradurally.

Extradural lipomas have been found, and although they are rare except when they are associated with spina bifida, they generally are found in the lumbar region.

Myeloma is, or multiple myelomas are, the rarest of the four types of primary malignant tumors of bone. The lesions constitute a disease of the bone marrow.

Neurofibromas occur extradurally. They belong to the same group as does the perineural fibroblastoma of Penfield. These tumors may occur extradurally or intradurally; they have even occurred in the intramedullary situation. Many neurofibromas within the spinal canal have a tendency to degenerate or become cystic;
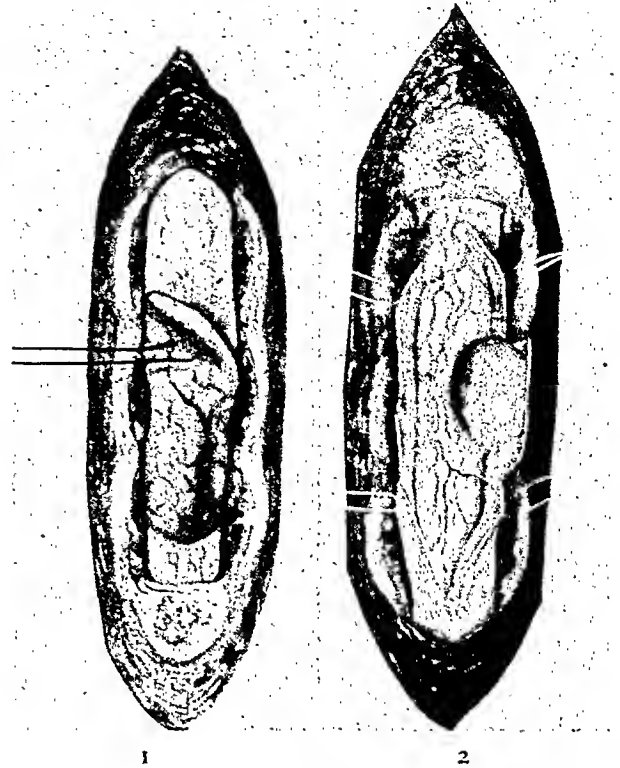
FIG. 1. Extradural tumor. Such a tumor develops from tissues present in the extradural space; retouched photograph.

FIG. 2. Intradural extramedullary meningioma or endothelioma; retouched photograph.

in a number of instances in the lumbar region neurofibromas have been known to grow to considerable size, eroding the laminas, pedicles and bodies of the vertebrae without producing signs of complete paraplegia. Neurofibromas also produce extradural, intradural and extravertebral tumors, the so-called hourglass or dumbbell perineural fibroblastomas. These may be discovered as tumors within the wall of the thorax, and only on careful examination can any signs of pressure of the spinal cord be elicited. These tumors should be removed very carefully; the intrathoracic mass ought to be removed in conjunction with the intraspinal mass to guard against intraspinal hemorrhages which have occurred to complicate such operations.

INTRADURAL EXTRAMEDULLARY LESIONS

It is interesting to observe that the intradural extramedullary types of tumors comprise approximately half of the entire group of tumors of the spinal cord. Fortunately, it is possible to remove most of
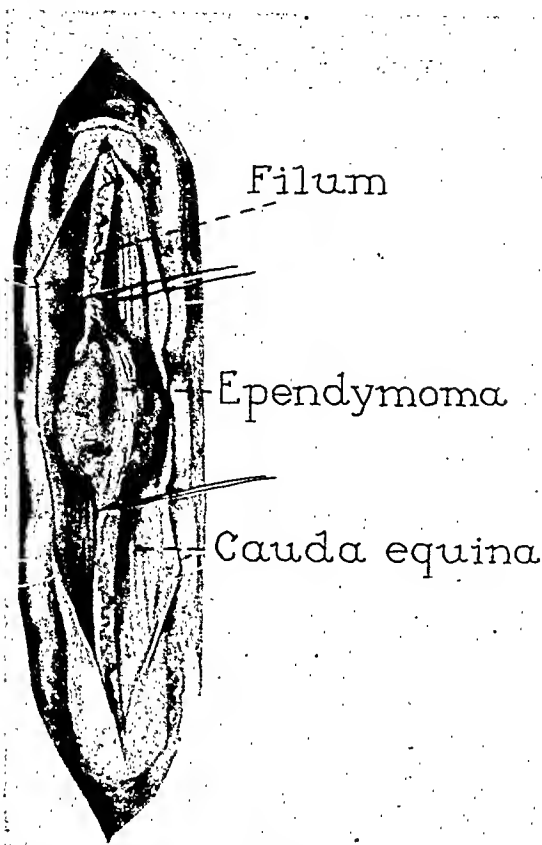
FIG. 3. Ependymoma occurring on the filum terminale producing compression on the cauda equina; retouched photograph.

FIG. 4. Neurofibroma, which may occur either intradurally or extradurally or may be combined as illustrated, showing method of compression on the cauda equina; retouched photograph.

FIG. 5. Intramedullary tumor, showing method of exposure and removal; retouched photograph.

them at operation, and most of them are benign and do not have a tendency to recur.

The so-called endotheliomas (Fig. 2) are found predominantly in intradural but extramedullary situations. They form the largest single class of tumors of the spinal cord. Fortunately, in view of their numerical incidence, they are associated with an excellent prognosis postoperatively. Although they are benign and encapsulated, these tumors have a tendency to recur if their dural attachment is not removed at the time of operation.

Ependymomas (Fig. 3) are tumors of the spinal cord found attached to the fibers of the cauda equina. They have proved to be tremendously difficult to classify pathologically.

Fibromas and fibrosarcomas may occur within the dura and involve the spinal cord only by compression. They can be completely removed at operation.

As a cause of compression of the spinal cord, intradural neurofibromas (Fig. 4) were found to be second, in point of number, only to the meningiomas or meningeal fibroblastomas.

INTRAMEDULLARY LESIONS

Thus far consideration has been given herein to extramedullary tumors which produce symptoms by the exertion of compression on the spinal cord. A third group consists of those lesions which involve the spinal cord by growing within its substance. (Fig. 5.) Primary tumors of the cord predominate, but occasionally a benign encapsulated tumor is found which proves to be a neurofibroma, an endothelioma, a lipoma or a fibroma. Moreover, there is always the possibility that there is a metastatic lesion within the cord which proves to be either carcinoma or sarcoma.

It has been found that of the primary tumors of the spinal cord and filum terminale, by far the most common are the ependymomas. However, the types of tumor which are found in the brain also

may be found in the spinal cord. Kernohan has found astrocytomas, astroblastomas, spongioblastomas, oligodendrogliomas and oligodendroblastomas, ganglioneuromas or neuroblastomas in the spinal cord.

SUMMARY AND COMMENT

Intraspinal tumors as a cause of pain and disability have been overlooked in recent years because of the discovery that many of the heretofore unexplained pains in the back and upper and lower extremities actually are the results of protruded intervertebral disks.

Clinically, the majority of intraspinal tumors are benign and can be removed. The possibility of metastatic lesions must be kept in mind, but the age of the patient and a careful general examination will rule this out. Degenerative lesions of the spinal cord also must be ruled out before a definite diagnosis can be made. Careful neurologic examination, including attention to both sensory and motor areas, should be carried out in all cases. Roentgenologic examination of the vertebral column often is of value in differential diagnosis. The introduction of iodized oil within the subarachnoid space has been of immense value in the localization of intraspinal tumors before they have progressed to the point of compression at which definite sensory levels are produced.

Intraspinal operations can be carried out with a minimum of danger. The majority of intraspinal tumors are benign and removable.

Anatomically, intraspinal tumors can be classed as extradural, intradural and intramedullary. Fortunately, the majority of tumors occur outside of the spinal cord, and the symptoms are produced by pressure. Surgical removal of this compression of the spinal cord results in relief of the clinical symptoms. The cardinal symptom of intraspinal tumors is pain, pain which appears intermittently and is increased by coughing and sneezing, pain which occurs after the patient is lying in the prone position, pain which usually causes the patient to arise early in the morning and sit in a chair, and which as a rule is confined to certain dermatomes, indicating the compression of one or more spinal nerves. Compression of the spinal cord produces symptoms, both sensory and motor; generally the so-called Brown-Séquard syndrome is present, with motor symptoms on the ipsilateral side and sensory symptoms on the contralateral side.

It is sometimes difficult to distinguish intramedullary tumors of the spinal cord from tumors of the extramedullary type, although early involvement of the vesical and rectal sphincters as a rule is indicative of an intramedullary type of tumor.

On the whole, it can be said that intraspinal tumors are removable, benign and do not tend to recur. If such a tumor is removed before irreparable changes have taken place in the spinal cord as a result of compression, complete restoration of motor and sensory loss below the level of the lesion should take place.

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TRAUMATIC INTRACRANIAL HEMORRHAGE*

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INTRACRANIAL hemorrhagic collections resulting from injury have been classified upon the basis of an anatomic location within the cranial cavity. The main groups include: (1) Epidural hemorrhage, produced by tearing of the middle meningeal vessels or dural sinuses, (2) subdural collections resulting mainly from disruption of cortical pial vessels, (3) subarachnoid hemorrhage produced by cerebral contusion and laceration and (4) intraparenchymatous hemorrhage due to intracerebral bleeding.

The type of hemorrhage may be correlated to some degree with the nature of the injuring force. When a direct blow or force strikes the non-moving or slower moving head, localized vascular injury is produced, the middle meningeal vessels may be torn, a venous sinus disrupted or a pial vessel ruptured as cerebral tissue is bruised and lacerated. The forces of indirect injury, when the head is decelerated as it rapidly moves against a slower moving or non-moving object, produce more complex, diffuse and combinations of vascular damage. The greater the velocity of the energy involved, the more extensive the pattern of vascular disruption.

The frequency of combinations of both vascular and parenchymatous damage must be emphasized since both the diagnosis and management are influenced by this circumstance. Thus, an extradural hemorrhage may co-exist with cerebral contusions, intracerebral petechial hemorrhages, subarachnoid and subdural bleeding. (Fig. 1.) However, one lesion is usually predominant and its clinical-surgical characteristics are sufficiently typical to warrant separate classification and discussion.

The following classification of vascular lesions is used in this analysis with a review of the pertinent findings in surgically treated and autopsied cases.

1. Epidural hemorrhage
2. Subdural hemorrhage
    (a) Acute type
    (b) Subacute chronic type
    (c) Acute and chronic types in infants
3. Subarachnoid hemorrhage
4. Intraparenchymatous hemorrhage
    (a) Petechial
    (b) Massive
5. Subdural accumulation of spinal fluids

EPIDURAL HEMORRHAGE

The most common type of extradural hemorrhage is of middle meningeal vessel origin. (Table 1.) Epidural hemorrhage from the sagittal sinus is occasionally seen. The latter may occur from depressed, comminuted fractures near the midline of the vault. In one of the cases studied an ice pick perforated the sinus, causing extensive extradural and intradural hemorrhage. A large clot collected between the two hemispheres in this instance and overlay the corpus collosum. Extradural hemorrhage of occipital emissary vein origin was noted in several cases of depressed fracture posterior and superior to the mastoid region. Occasionally the lateral sinus was involved, an occurrence associated with penetrating wounds.

Since extradural hemorrhage is usually of middle meningeal origin, the terms middle meningeal, epidural and extradural hemorrhage are used interchangeably. This type of collection is usually unilateral. Two cases of bilateral extradural clots have been

seen in this series. One patient was operated upon and the other was seen at the post-mortem table.

Etiology. Falls, bicycle accidents and direct head blows are frequently the cause of extradural hemorrhage. In a series of 158

TABLE 1

THIRTY-FIVE OPERATED CASES OF MIDDLE MENINGEAL HEMORRHAGE

State of consciousness	
Lucid interval......................	13
Unconscious throughout..............	13
Drowsy and disoriented..............	8
Short unconsciousness followed by normal conscious state................	1
Pupils	
Larger on same side.................	25
Larger on opposite side..............	1
Equal.............................	9
Extraocular palsy	
Third.............................	4
Fourth............................	1
Sixth.............................	1
Focal signs	
Present...........................	30
Not present.......................	5
Vital functions	
Pulse.............................	45–60
Respirations.......................	18–26
Temperature.......................	100–102
Spinal-fluid findings	
Pressure..........................	45–650
Bloody............................	In 17 cases
Clear.............................	In 1 case
Associated massive lesions	
Subdural hemorrhage................	7
Temporo-sphenoidal-lobe clot........	4
Subdural accumulation of spinal fluid...	2
Recovered.....................	26
Died..........................	9

autopsied cases of cranial injuries, seven out of eleven fatal cases of epidural hemorrhage were the result of such low velocity, direct injuries. High velocity automobile injuries involving indirect forces appear to cause a smaller number of middle meningeal hemorrhages.

Clinical Observations. Although the classic "lucid interval" characteristic of the clinical syndrome of extradural hemorrhage may occur, it is the exception rather than the rule. An already unconscious state gradually deepening is commonly observed. A "lucid interval" may be absent due to co-existing pathologic damage within the cranial cavity. Occasionally hemorrhage from the meningeal vessels superimposed

upon a concussive state may be so rapid as to preclude a conscious period.

A frequent clinical finding is dilatation of the pupil on the side of the lesion. Extraocular palsies occur. The enlarging clot may compress the ocular nerve or nerves as they traverse the superior orbital fissure or the pressure may occur by the bulging inner aspect of the temporal lobe medially, involving and interrupting the nerve or nerves in their intracranial course from brain stem to the cavernous sinus. A dilated pupil without other signs of oculomotor paralysis may also be attributed to the above described etiology or to paralysis of the cortical pupillary constricting mechanism. In favor of a peripheral mechanism is the fact that when a clot is located at the base, pupillary manifestations are the rule. (Fig. 2.) When the clot covers the parietal and temporal areas, the pupils are likely to be equal in size. (Fig. 3.) A dilated pupil is usually on the side of the lesion. A constricted pupil on the side of the lesion was seen only once in this series. Paralysis of the third nerve was observed in four cases.

Increasing weakness and paralysis result from compression of the clot over the motor centers. The paralysis is usually on the opposite side from the clot. Occasionally it may be seen on the same side as the clot. In the untreated case the patient fails rapidly; the deep tendon reflexes become diminished, then lost; the plantar reflexes become unobtainable; the corneal response becomes imperceptible; generalized or Jacksonian convulsions may occur. The importance of constant observation cannot be overemphasized since these changes may follow one another in rapid succession with eventual fatal outcome.

Roentgen Observations. Survey roentgen studies of the skull may be of assistance in the diagnosis of an extradural hematoma. A fracture line crossing the temporoparietal region suggests this possibility. (Fig. 4.) Depressed temporal fractures may be associated with hemorrhage; five cases occurred in this series. Because of this association, patients with simple depressions, even if
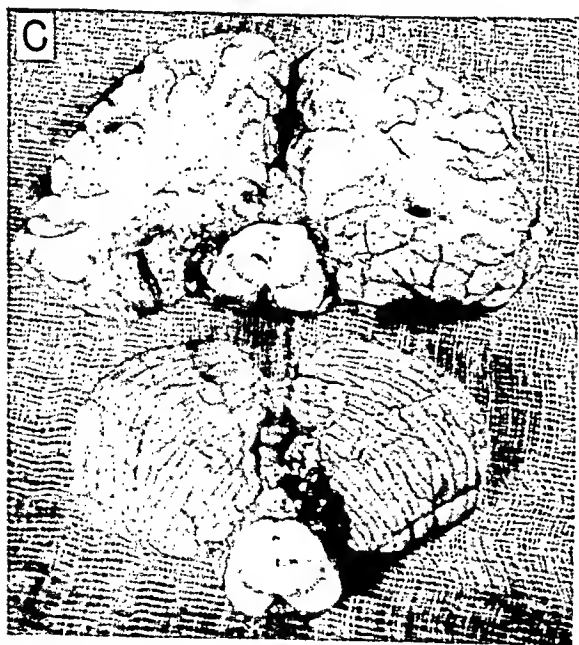
FIG. 1. Autopsy findings in a case with right epidural hematoma (A); subarachnoid hemorrhage (B); mid-brain hemorrhages and contusion of right occipital lobe (C); hemorrhages in the cerebellum and pons (D). That a combination of pathologic conditions co-exist is well known. However, usually a single pathologic entity predominates, producing fairly typical signs and symptoms.
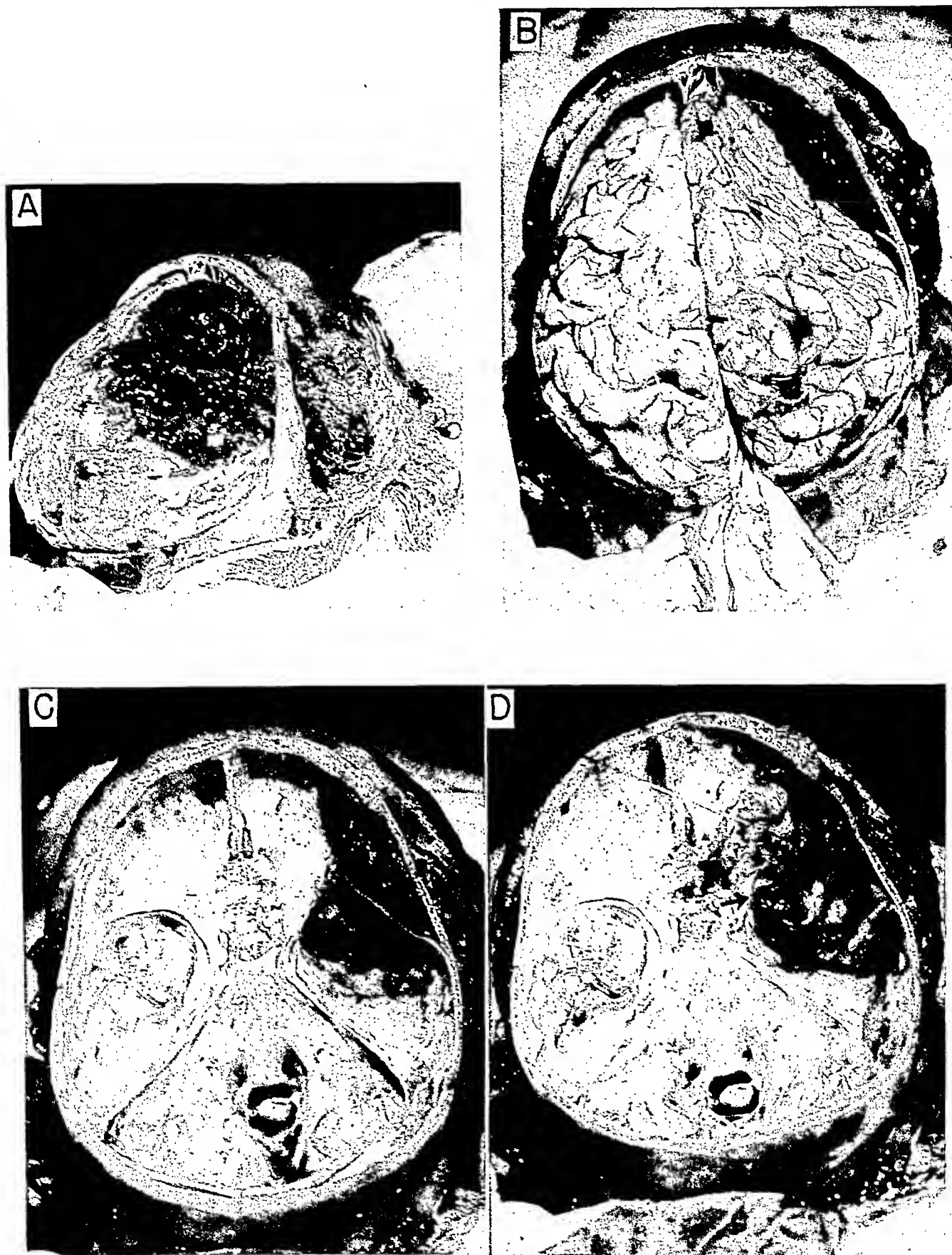
FIG. 2. Epidural hematoma over the convexity (A) causing marked compression of brain (B), dissecting its way into the middle fossa all the way to the region of the superior orbital fissure (C), and dissection showing the extent of the hemorrhage in the superior orbital fissure (D). This explains the usual involvement of the third nerve by compression in its course through the orbital fissure resulting in widely dilated pupil or ocular motor palsy.
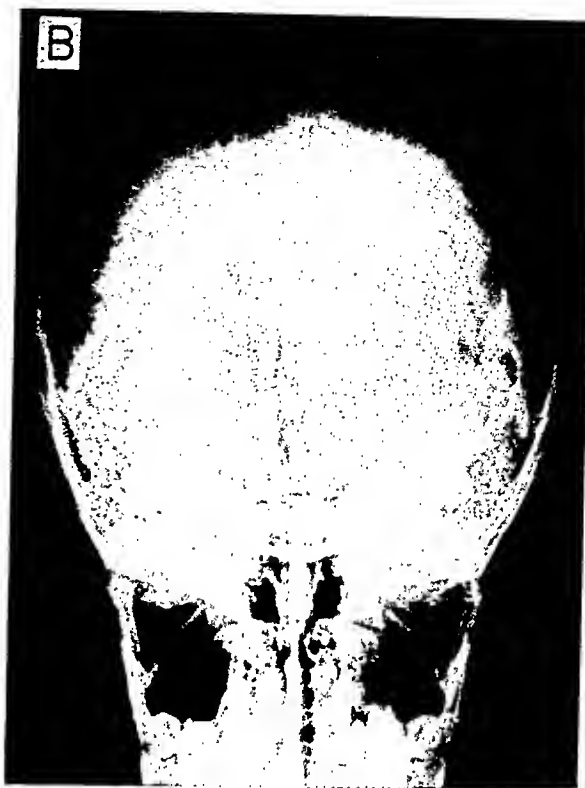
FIG. 3. A and B, encephalogram showing compression of right ventricle from epidural hematoma; hemorrhage only over the convexity with no inequality of pupils.

minor, deserve close observation, particularly if continued unconsciousness and focal signs exist. In two cases, demonstrated by postmortem examination, the fractures were of such degree that roentgen studies could not have revealed them. In one case of bilateral middle meningeal hemorrhage both temporal areas were explored because of the extensive fracture from one temple to the other. The patient presented bilateral neurologic signs. In patients with a pineal calcification, the presence of a shift may be diagnostic and may locate the lesion accurately. (Fig. 7.)

Pathologic Observations. The initial hemorrhage from the middle meningeal vessels may be instantaneous and rapid or delayed and slowly progressive. In progressive lesions, various venous channels of the dura and bone contribute as the separation of the dura from the bone proceeds. Accompanying subdural bleeding was noted in eleven out of thirty cases. In two patients there was an associated subdural accumulation of spinal fluid. In three instances, a massive temporosphenoidal clot was found to accompany the extradural collection.

Treatment. In our experience the operation of choice is that of a subtemporal decompression on the side of the lesion. If the clot is beyond the area of the temporal bone, a small bone flap may be required. Bleeding points are controlled. The middle meningeal artery is ligated or occluded by silver clips or cautery. Deep hemorrhage at the base may require retraction of the temporal lobe for exposure of the foramen spinosum into which a plug of cotton or wood may be introduced. The dura is opened, if necessary, to remove a subdural or intraparenchymatous clot. Usually a gauze drain is used for hemostasis. Local or general anesthesia may be used. In our experience pentothal sodium has been dangerous.

ACUTE SUBDURAL HEMORRHAGE

Acute subdural hemorrhage usually results from a tear of pial vessels associated with contusion and laceration of cerebral

FIG. 4. A and B, epidural hematoma in a patient with a linear fracture of the left parietal bone extending into the middle fossa.

tissue. (Table II.) At times it is extensive, covering the surface of a hemisphere. Bilateral collections may be present. As a rule, the hematoma is located in the fronto-parieto-temporal region of one or both sides. Occasionally an unusual localization

TABLE II
ACUTE SUBDURAL HEMORRHAGE

Lucid interval.	8
Continued unconsciousness with disorientation.	19
Dilated pupil. . . . .·.	17
Hemiparesis or hemiplegia.	20
Dilated pupil with contralateral paralysis or paresis.	13
Convulsions (Jacksonian).	6
Convulsions (generalized).	4
Total cases.	39
Deaths.	15

of the clot is seen. (Fig. 5.) In two patients there was an extensive subdural hematoma between the two hemispheres.

The common fronto-parieto-temporal clot site can be explained on the basis of anatomic relations between brain and bone. The frontal and temporal lobes are snugly fitted into a bony encasement having projecting and irregular surfaces. In indirect blows when the rapidly moving head strikes a non-moving or slowly moving object, the intracranial contents by a mass movement abut against the irregular surfaces with resulting vascular disruption in this region.

Clinical Observations. The conscious state may manifest changes which correspond with those observed in patients with extradural hemorrhage. The "lucid interval" is common and is seen as frequently as in middle meningeal hemorrhage. The interval may vary from a few hours to several days or weeks. Since expansion of the subdural lesion occurs more slowly than the epidural, resulting physiologic events are usually less prompt in appearance. The longer the period of lucidity and freedom from major symptoms, the better the prognosis. Patients may remain semiconscious or comatose from the time of injury. Focal neurologic signs may be meager, those present being the result of increased intracranial pressure. Frequent examinations may reveal a gradually deepening stupor

with a dulling of responses to stimuli. A slowing of the pulse may occur. Occasionally the blood pressure rises with increasing stupor but as a rule it shows little or no change. Convulsions are common. Pupillary inequality and extraocular palsies occur. A dilated pupil, if present, is usually on the same side as the lesion. Unilateral extraocular muscle paralyses are also usually ipsilateral. There is most commonly an absence of papilledema. Increasing paresis of one-half of the body may be seen. This paresis or paralysis may be on the same side as the subdural collection, resulting in false localization. The spinal fluid pressure is usually elevated but surprisingly low pressures have been noted. The cerebrospinal fluid is often bloody but may be xanthochromatic or clear. Severe bloodly spinal fluid most frequently indicates diffuse intracerebral injury but exceptions to this conclusion are commonly encountered. Failure of a patient who has sustained a cranial injury to improve normally suggests the possibility of an existing complication which may well be a subdural collection. By means of pneumoencephalography or multiple burr holes the diagnosis may be made.

Roentgen Observations. A fracture of the skull may be present on the same or on the side opposite the hematoma with equal frequency. (Fig. 6.) A fracture is commonly absent. The presence of a fracture line does not locate the collection in cases of acute subdural hemorrhage. The presence of a shifted pineal shadow may be diagnostic. (Fig. 7.)

Pathologic Observations. As a rule, the disruption of pial vessels is the source of a subdural hemorrhage. Occasionally a massive intraparenchymatous clot may extrude through the cortex and seep into the subdural space. The latter is more common in non-traumatic cases. Contusion and laceration of the cerebral substance may accompany pial tears; deeper multiple small hemorrhages may be seen. These associated lesions complicate the syndrome of the subdural collection and serve as a cause for
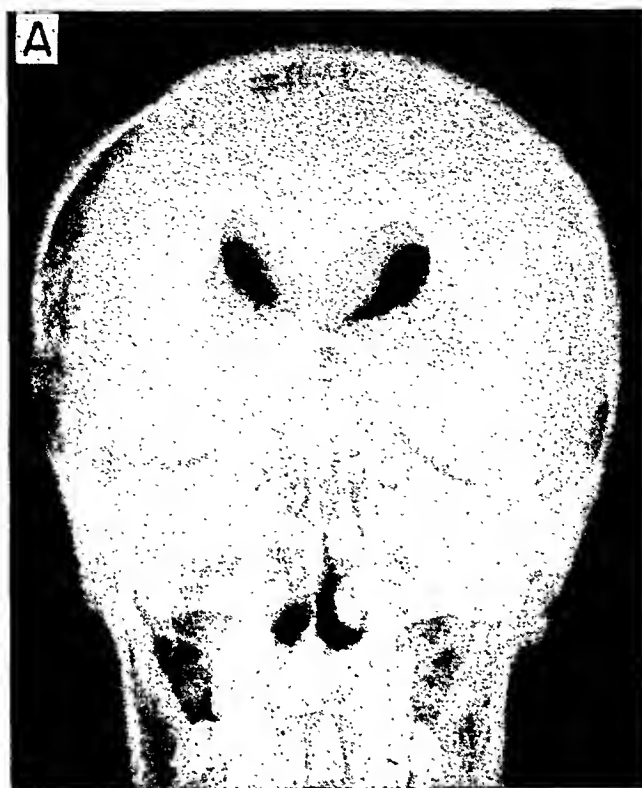
FIG. 5. A and B, unusual localization of subdural hematoma. Air studies suggest a large mass between the two hemispheres which was a subdural hemorrhage from tear of connecting veins between cerebrum and superior sagittal sinus.

FIG. 7. Pineal shift in a patient with acute subdural hematoma.

FIG. 6. Subdural hemorrhage (A) in a case with a fracture on the same side as the hemorrhage; (B) in a patient with a fracture on the opposite side; (C) in a patient with no fracture.

toms. In the slowly expanding forms, the absorption of tissue fluids and cerebrospinal fluid by the collection may only gradually add volume and only insidiously result in a dynamic intracranial condition. This process becomes characterized by its chronicity and is so described by the term chronic subdural hematoma. In instances in which the localizing signs are on the wrong side there probably occurs compression of the brain stem on the opposite side from the hemorrhage against the tentorial border, compressing the pyramidal tract on the opposite side.

Treatment. The acute subdural hemorrhage presenting dynamic signs usually necessitates operative intervention. Both hemispheric surfaces should always be explored. A trephine opening at the fronto-parietal junction 2 inches on either side of the midline uncovers the greatest majority of collections. If the two initial openings fail to reveal a hematoma, an air study may be employed in place of further random openings. If a subdural hemorrhage has been located, a subtemporal decompression

prolonged disability or death. A subdural hemorrhage may be of non-surgical proportions producing minor or no symptoms and signs. Upon the initial size and source of the hemorrhage depends the expanding nature of the mass with slow or rapid progression of symptoms and signs. A disruption of a large pial vein may produce practically instantaneous signs and symp-
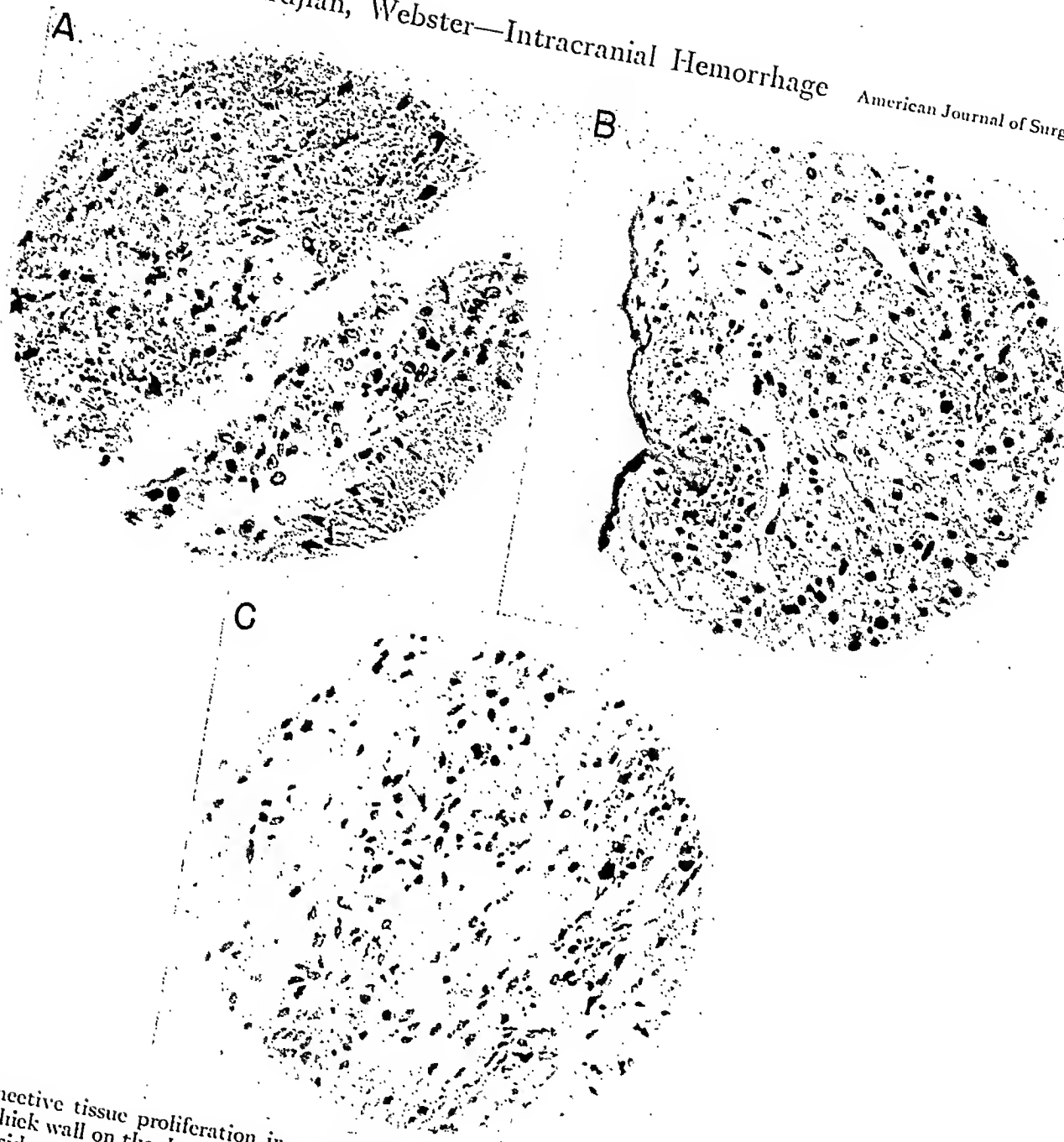
FIG. 8. Connective tissue proliferation in a patient with acute subdural hemorrhage eighteen hours after injury (A). Thick wall on the dural side of chronic subdural hematoma six weeks after injury (B). Thick wall on the dural side of chronic subdural hematoma several months after injury (C).

is then performed on the side of the lesion. The liquid and/or semi-solid clots are irrigated from the subdural space by means of saline and a brain spatula. The subdural space is drained from twelve to eighteen hours.

SUBACUTE AND CHRONIC SUBDURAL HEMATOMAS

When hemorrhage into the subdural space is small in amount or of quantity. which is not sufficient to jeopardize the spacial relationships and requirements

within the cranial cavity, symptoms may be absent for weeks or months. By osmosis and the diffusion of cerebrospinal fluid and other tissue fluids into the collection (having a higher specific gravity), the subdural hematoma gradually expands. Additional volume is contributed by occasional hemorrhages into the colt from vessels in the proliferating granulation tissue on the dural side of the collection. (Fig. 8.) Ultimately the expansion results in cerebral symptoms. The prolonged, lucid, symptom-free interval can be explained upon these circumstances. (Table III.)

Clinical Observations. Chronic subdural hematoma may follow minor types of cranial injury. In some instances the trauma may be entirely forgotten by the patient and relatives. On the other hand, severe cerebral damage may be accom-

TABLE III

SIXTY-FIVE OPERATED CASES OF CHRONIC SUBDURAL HEMATOMA (ADULT)

Headaches
  Present over the lesion........................ 42
  Not complained of, or no history.............. 12
  Generalized................................... 11
Pupils
  Dilated on side of lesion..................... 13
  Equal... 49
  Dilated on side opposite lesion............... 3
Extraocular palsy
  Third... 4
  None.. 61
Fracture of skull
  Present....................................... 21
  None.. 44
Side involved
  Left.. 32
  Right... 27
  Bilateral..................................... 6
Spinal fluid findings
  Pressure above 300............................ 40
    200–300..................................... 12
    100–200..................................... 5
  Blood or xanthochromatic......................
Operation of choice:
  Bilateral, exploratory, trephine subdural temporal decompression on the side of lesion or osteoplastic flap if clot is solid
    Recovered.................................... 59
    Died... 6

panied and complicated by this condition. In such cases there may be fracture of the skull with disability from the onset of the injury.

If chronic subdural hematoma complicates a severe injury, the patient may remain unconscious or semi-conscious for varying periods from days to weeks, progressing from the unconsciousness caused by the initial severe cerebral damage into a semi-consciousness caused by the cerebral compression of the subdural hematoma. A number of patients in this series had associated severe, diffuse cerebral injury.

In the more typical case the patient may be normal following a cranial injury for varying periods. Then in a brief period of but several days headache, previously of mild order, becomes intense. Conduct and personality defects appear and within a week the patient progresses into a stupor, and finally coma if recognition of this complication has not been made.

Pupillary inequality may occur with the enlarged pupil on the ipsilateral side. The fundi may exhibit papilledema although this is the exception rather than the rule. Visual field studies in those patients who are cooperative have proved of little help. Headache is a constant complaint and may be localized to the side of the lesion. An overlying hyperpathic zone of the scalp may be present. In some patients increasing drowsiness may be the only finding. As a general rule, focal neurologic signs obtain in the advanced cases. Bilateral focal signs and findings implicating the "wrong side" are common. This is the result of peduncular compression against the tentorium on the side opposite the lesion. The cerebrospinal fluid pressure is usually elevated. The fluid itself may be clear but is frequently xanthochromatic. The grossly clear fluid may contain an elevated total protein with minimal pleocytosis.

Changes in pulse, respiration, blood pressure and temperature are of little diagnostic significance until advanced cerebral compression has occurred. At times a bradycardia may be observed usually accompanied by headache. The electroencephalogram may be of important diagnostic assistance. (Figs. 9 and 11.)

Roentgen Observations. A fracture of the skull may or may not be present and its presence does not indicate the side of the hematoma. As a rule, the complication follows minor trauma to the head and thus a majority of patients show no fracture. Pneumoencephalograms are very helpful and patients with subdural hematomas tolerate this procedure surprisingly well. If the ventricles are visualized, there is a midline shift and cortical air markings on the affected side are characteristically absent. (Figs. 9 and 10.) A collection of air may show medial to the hematoma and this column may appear as a "pointer" within the cranial cavity. This finding is pathog-
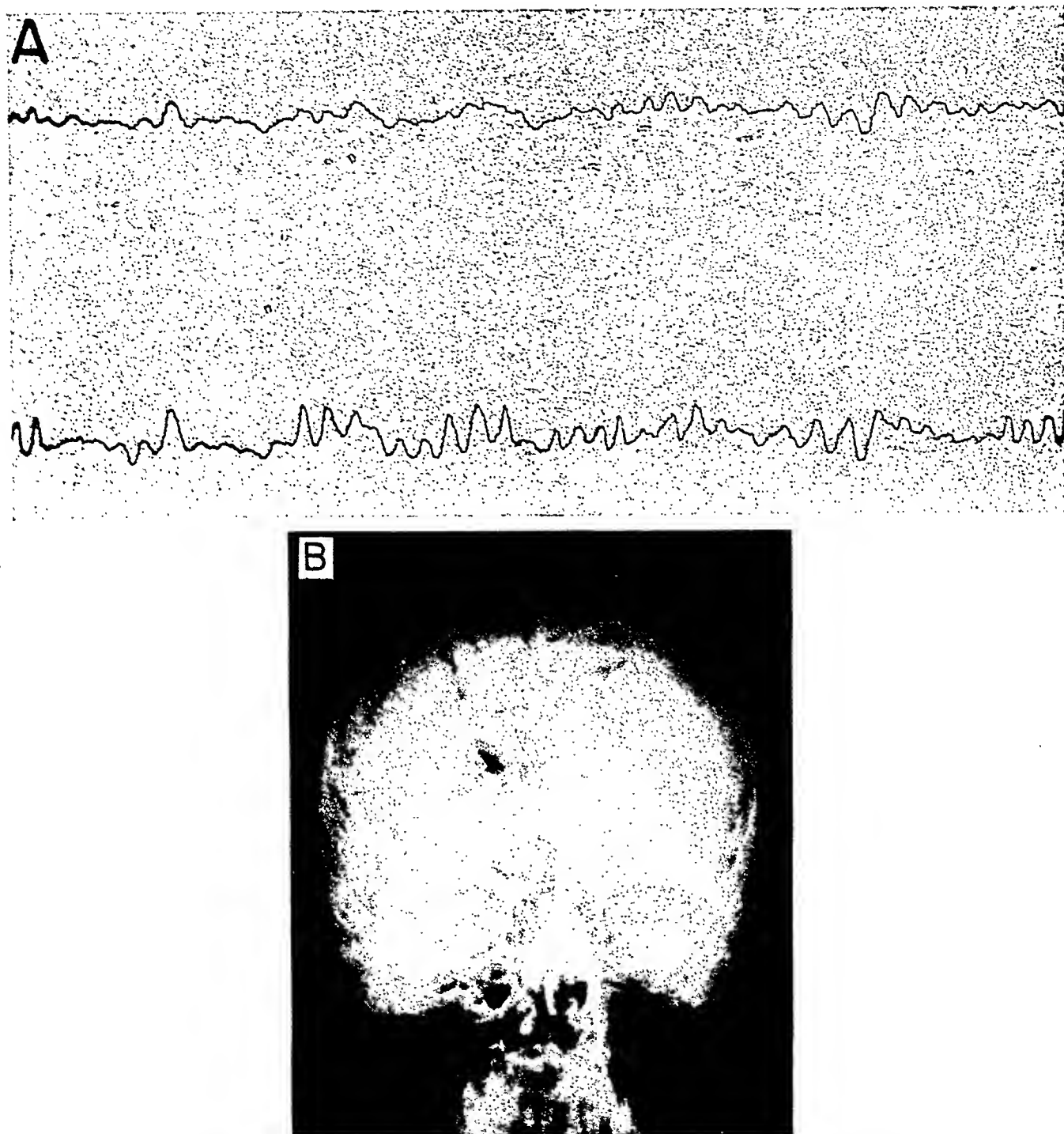
FIG. 9. A and B, air study and electroencephalogram in a patient with right subdural hematoma. Note absence of subarachnoid spaces on the affected side. Electroencephalographic record with disorganization of the electrical pattern on the affected side.

nomonic of a subdural hematoma. Pineal shadow shift may be diagnostic of the location of the lesion. (Fig. 7.) Electroencephalographic studies may be diagnostic (Fig. 11), although in our experience correct localization of the lesion has been accomplished by this technic in about 40 per cent of the cases.

Pathologic Observations. In the genesis of chronic subdural hematoma there is a prompt cellular reaction with connective tissue proliferation on the dural side of the collection. (Fig. 8.) On the arachnoid side a limiting membrane forms. The subdural mass increases in size by the absorption of tissue fluids and spinal fluid through the process of osmosis and diffusion. The inner layer of the sac serves as a semi-permeable membrane. Thus an expanding process occurs (usually covering a wide area of the

this tissue to retain its characteristics of a semi-permeable membrane is lost.

SUBDURAL HEMATOMA (ACUTE AND CHRONIC) IN INFANTS

Newborn infants occasionally are afflicted with subdural hemorrhage. Birth injuries, injury by forceps, falls and other etiologic factors may be responsible. In acute subdural hemorrhage, the infant does not react normally to its environment, presenting often a lethargy and dulled response to stimuli. A weak, inconstant cry replaces the lusty ability of the normal infant. Cyanosis appears with weak, irregular respirations. High concentrations of oxygen may have little effect. Neurologic findings may be of a minor order. Occasionally spasticity is present on the contralateral side. Pupillary irregularities occur. Convulsions are common, being either localized or generalized. An important finding is a tenseness, fullness or bogginess of the anterior fontanel. Lumbar puncture usually shows a grossly bloody fluid under increased pressure.

The infant with a chronic type of subdural hemorrhage or hydroma has progressed satisfactorily in its early months until a parent or pediatrician notices enlargement of the head. In other patients, a convulsion first draws attention to an interruption of normal progress. In all other respects, the growth, sleep, feeding, vision, etc., may be normal. Head measurements indicate an enlargement above normal. A cracked pot sound may be noted on percussion of the head. The fontanel is wide and the sutures may be separated. Roentgen studies may show this same suture separation. Focal neurologic signs and fundus changes are usually absent.

The lesion may be accurately diagnosed by tap with a No. 18 gauge needle through the lateral aspect of the fontanel on both sides. When present, the collection may be evacuated through appropriate trephine openings on the affected side. Occasionally aspiration alone has been sufficient to drain the collection.

FIG. 10. Marked compression of the brain with ventricular distortion in a case of acute subdural hematoma.

cerebral surface, since the subdural space is not limited) rather than an absorption of the collection. Within the sac of the hematoma the blood may partially or completely hemolyze. It is not unusual, however, for the clot to remain semi-solid.

Treatment. Bilateral trepanation at the frontoparietal junction about 2 inches on either side of the midline followed by a subtemporal decompression on the side of the collection is the method of choice if the lesion is mostly fluid. A small osteoplastic flap is used if the clot is mostly solid. A single or two trephine openings may be adequate for the evacuation of unclotted hematomas when combined with irrigation. Changing the position of the head may also be of assistance. It is now generally admitted that the membrane of the hematoma sac need not be removed. After evacuation of the sac contents the ability of

FIG. 11. Electroencephalographic records in two cases of subdural hematoma. In (A) there is disorganization of the electrical pattern over the right temporal region. In (B) there is delta activity over the left hemisphere.
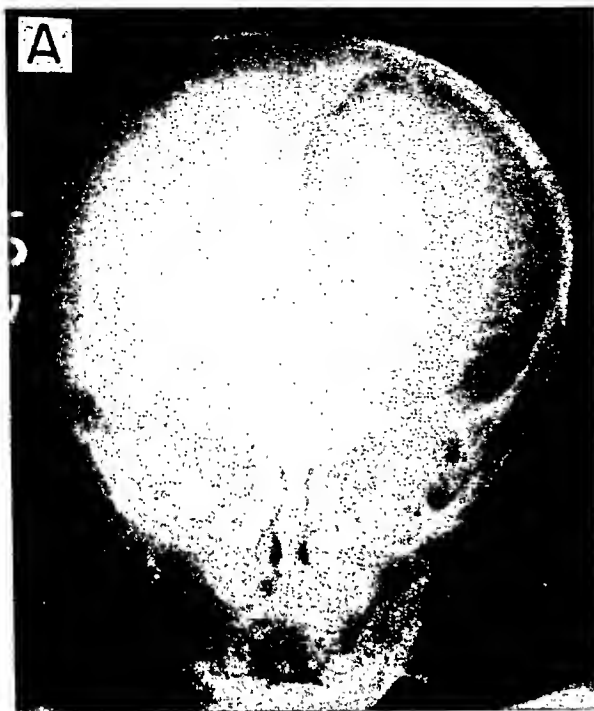
FIG. 12. A and B, subdural hematoma in a four months old infant visualized by injecting air after drainage of fluid contents. Note that the entire hemisphere was compressed by the fluid mass.

The chronic subdural collection is suitably managed by means of two fronto-parietal openings of an exploratory type. If a clot is encountered with a thick membrane, it may be necessary by means of a small bone flap to remove this layer in order to allow expansion of the depressed hemisphere. (Fig. 12.) Clots may be irrigated from the surface through two openings. A hydroma may be evacuated through a single opening.

SUBDURAL ACCUMULATION OF SPINAL FLUID

Consideration must be given to the occurrence of a subdural accumulation of

TABLE IV
SUBDURAL COLLECTION OF SPINAL FLUID

Lucid interval	3
Continued unconsciousness with disorientation	16
Dilated pupil	9
Hemiparesis or hemiplegia	8
Dilated pupil with contralateral paralysis or paresis	5
Convulsions (Jacksonian)	2
Convulsions (generalized)	2
Total Cases	20
Deaths	-

spinal fluid. (Table IV.) This curious condition is seen in less severe grades of head injury. It is almost never diagnosed prior to operation. That there is a cause and effect relationship between this condition and clinical findings is proved by the rather remarkable results following treatment in some cases. Pathologically, the collection has been stated to be caused by a rent in the arachnoid with a resulting valve-like action which permits cerebrospinal fluid to enter the subdural space and not return. When a patient fails to improve after drainage of a collection, air studies may be necessary to rule out a co-existent disturbance. We have seen middle meningeal hemorrhage occur with this lesion. In some cases this abnormality may be seen without a history of cranial injury.

The treatment is that of simple drainage of the subdural collection through trephine openings. Not infrequently the collections are bilateral.

MASSIVE TYPE INTRAPARENCHYMATOUS HEMORRHAGE

Massive intraparenchymatous hemorrhage or intracerebral hemorrhage in acute head injury is uncommon in autopsy ma-

terial. The most vulnerable area seems to be in the region of the temporosphenoidal lobe. Clinically the same rarity of such cases is evident. In our series of over 200 operations for head injury with massive hemorrhage, there have been five instances of massive intracerebral clot; three occurred in the left temporosphenoidal lobe.

The clinical findings observed were similar in each instance to left temporosphenoidal clot. A progressive failure characterized by a right lower facial weakness, right upper limb paresis, eventual aphasia and stupor was noted. Because of the localizing signs, trephine with a left subtemporal decompression was performed and in each instance a massive clot from the temporosphenoidal lobe with an overlying subdural hemorrhage was evacuated. Much necrotic brain tissue was encountered which was also removed. (Fig. 13.)

In the diagnosis of such cases, localizing neurologic signs aided by air studies should accurately locate the lesion.

FIG. 13. Extensive contusion of brain in right parietal region. Such a lesion may eventually result in softening and hemorrhages causing an intraparenchymatous mass lesion. Traumatic intraparenchymatous hemorrhage is almost always associated with softening and necrosis of brain tissue resulting in severe cerebral deficit.

SUBARACHNOID HEMORRHAGE

Subarachnoid hemorrhage is the most common variety of traumatic intracranial hemorrhage. It may co-exist with epidural, subdural and intraparenchymatous hemorrhage. The associated subarachnoid hemorrhage is usually brought about by laceration and contusions of brain surfaces.

TABLE V*

Dilated pupil	11
Hemiparesis or hemiplegia	15
Lucid interval	7
Continued unconsciousness or disorientation	26
Dilated pupil with contralateral paralysis	10
Convulsions (Jacksonian)	4
Convulsions (generalized)	3
Total cases	34
Deaths	16

* In thirty-four cases the above signs and symptoms led to exploration, but the latter revealed no massive clots. Usually edema of the brain, contusions and subarachnoid hemorrhage were seen.

At times it is extensive enough to cover one or both hemispheres. The diagnosis is confirmed by a lumbar puncture. The cerebrospinal fluid obtained from the spine may contain much less blood than that obtained from the cisterna magna or the ventricles. Localized subarachnoid hemorrhage with edema may cause focal signs of a progressive nature so as to resemble closely the picture of subdural hematoma or massive intracranial hemorrhage. The presence of blood in the spinal fluid should not deter operative intervention, for in cases of epidural and subdural hemorrhage there is frequently associated subarachnoid hemorrhage. The patient with subarachnoid hemorrhage usually gradually improves, the conscious or semi-conscious state lightens and focal signs resolve. Conservative supportive treatment is thus wisely continued in the presence of gradual improvement.

PETECHIAL HEMORRHAGE

Brief mention of petechial hemorrhages is made to complete the subject. How often they occur in those with severe cranial injury is difficult to establish, but 34 per cent of cases seen at the autopsy table show these small hemorrhages throughout the cerebral tissue. The association of petechial hemorrhages with other forms of operative

conditions makes for longer convalescence and less complete recovery due to sequelae of a permanent nature.

COMMENT AND SUMMARY

A similarity of symptoms and signs is evident in the discussion of the various types of acute traumatic intracranial hemorrhagic collections. The state of consciousness and its alterations are significantly important. An increasing stupor, abolition of the conscious state after a lucid interval are indications for possible operative intervention. The neurologic status of the patient should be studied frequently. The progress of neurologic findings may be suggestive of an enlarging mass lesion. On repeated examinations, if the patient shows increasing weakness of one-half of the body, eventuating in paralysis, he may be afflicted with an enlarging blood clot. On the other hand, if a paresis or paralysis has been found to exist soon after injury, its causation by contusions and bruises of cerebral tissue is much more likely and, therefore, operative intervention may not be considered in such a case. The presence of inequality of pupils may be significant. The dilated pupil is usually on the side of the lesion. Papillidema may be present although this is rare. The vital functions may show alterations from normal. A slowing pulse rate and a lowering respiratory rate may accompany mass lesions. An increase in blood pressure, although rare, may signify increasing intracranial tension.

In acute hemorrhagic collection the one single important symptom justifying exploration is increasing drowsiness or stupor. Pupillary inequalities, changes in vital functions and focal neurologic signs may corroborate the presence of an enlarging clot. However, a dilated pupil, a low pulse rate or hemiplegia in a perfectly conscious patient does not justify operative intervention.

In subacute and chronic subdural hematomas the presence of unilateral headache and hyperpathia of the scalp over the pathologic lesion are important. The association of severe brain injury with subdural hematoma produces long disability and the recognition of a complicating subdural hematoma in long continued unconsciousness is important.

Post-traumatic epilepsy is rare in epidural and chronic subdural hematomas. In acute subdural hematomas it is seen in almost a third of the patients. This favors the assumption that in these patients the subdural hematoma is associated with contusions and bruises of the cerebral surface eventuating in cerebrodural cicatrization. Headaches, dizzy spells and personality changes are surprisingly uncommon as sequelae in postoperative mass lesions.

SURGICAL TREATMENT OF TUMORS OF THE PITUITARY BODY*

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THE pituitary tumors observed by the senior author during a period of approximately fifteen years provide the material for this report. Histologic verification was not obtained in every case, a fairly large proportion of patients having been treated by means of irradiation exclusively. In the non-verified cases, the combination of clinical symptomatology and physical findings, together with the results of visual field studies and roentgen examination of the skull, established the diagnosis beyond reasonable doubt. Whenever the nature of the lesion was questionable, the issue was settled by operation. A few patients were seen only once or twice in consultation but sufficient pertinent data concerning them were made available to warrant including them in this study.

CHROMOPHOBE ADENOMAS

The chromophobe adenomas constituted the largest group of tumors, there being ninety-three cases in this series. The incidence of males was somewhat higher than that of females, the figures being fifty-two and forty-one, respectively. That the disease is primarily one of adults is indicated by the fact that 74 per cent of the patients were in the fourth, fifth or sixth decade of life at the time they sought medical attention. The fifth decade accounted for 34 per cent of all patients, the largest number in any comparable age group. Three patients were less than twenty years old.

Symptomatology. The most constant complaint was impairment of vision. It was either the chief symptom or one of the presenting complaints in eighty-six cases. Next in frequency were headaches which occurred in fifty-six patients. Disturbances referable to sexual function, amenorrhea or, in the male, loss of libido and potency, were present in fifty patients. Not infrequently the amenorrhea or impotence antedated the visual symptoms. Less often mentioned in the anamnesis were increasing obesity, a tendency toward somnolence, generalized weakness, diplopia and, in a few cases, polydipsia and polyuria. Mental symptoms were pronounced in six instances. Two of these patients presented unusual clinical pictures and in each case the diagnosis was resolved by pneumencephalographic examination, which revealed the tumor to be above the sella turcica instead of chiefly within it and projecting into the third ventricle. In both cases, the nature of the lesion was subsequently verified. A third patient proved to have a large intracranial extension into the middle fossa on one side. Mental symptoms and convulsions occurred in still another patient who had been operated upon twice previously, once twenty years ago and again thirteen years ago. Following the evacuation of a cystic tumor at a third operation, he improved considerably. He is still alive and continues to maintain his improvement. In a fifth case, recurrent growth of a tumor was associated with a manic state which disappeared after operation. This patient has been followed for a period of fourteen years and has remained free of any further recurrence. The remaining case was one in which headache, drowsiness and confusion supervened following a relatively minor head injury. A subtotal removal of the tumor was accomplished at operation but the patient expired postoper-
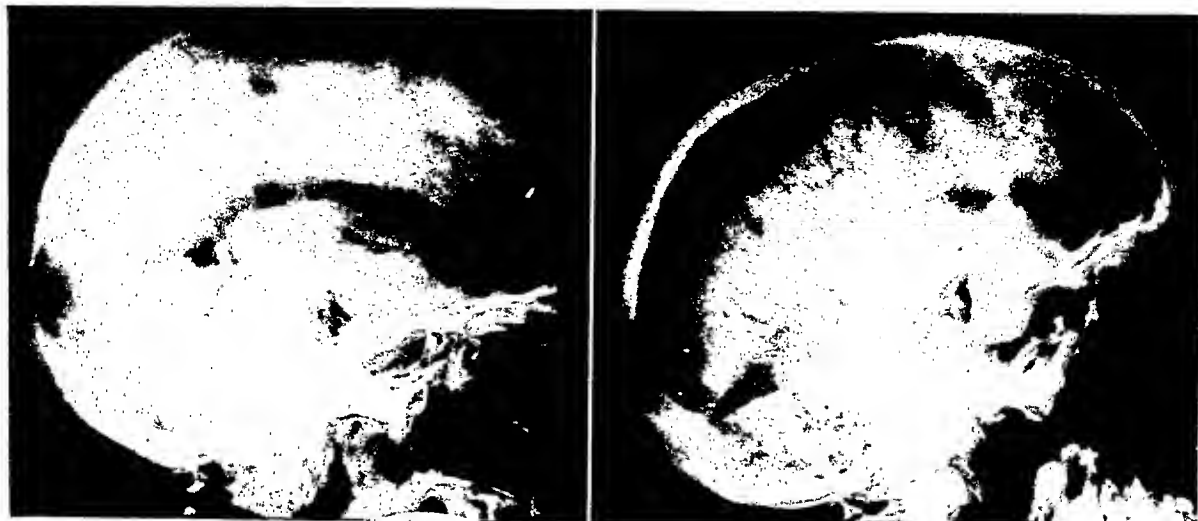
FIG. 1. Case 90. Pneumencephalogram following recurrence of symptoms and identical with original preoperative pneumencephalogram. Obliteration of anterior portion of cisterna interpeduncularis with concave defect rostrally.

FIG. 2. Case 67. Failure of visualization of the cisterna chiasmatis and of the cephalic half of the cisterna interpeduncularis. Note normal-sized sella turcica.

atively. A necropsy not having been performed, the extent of the lesion remains unverified.

The objective evidence supporting the diagnosis of a chromophobe adenoma was of a dual character. It consisted of constitutional stigmas resulting from diminished activity of the hypophysis, plus signs representing local pressure effects of the enlarging mass. Disturbance of sexual function, a cardinal manifestation of hypopituitarism, has already been mentioned and the frequency of its occurrence in this series noted. In fifty-four cases one or more of the following additional physical features were recorded: an unusual degree of pallor often described as a "waxy" or "pasty" complexion, dryness and atrophy of the skin, sparseness of the beard and a female escutcheon in males, a paucity of axillary and pubic hair, muscular flabbiness, obesity, and in men, a feminine type of fat distribution. Blood sugar levels were usually normal and sugar tolerance tests also within normal limits. The basal metabolic rate was almost uniformly diminished, averaging − 14 per cent. Roentgenographic evidence of an intrasellar tumor was present in all but three cases. The characteristic

findings included enlargement of the sella turcica with variable degrees of erosion of the posterior clinoids, dorsum sellae and sellar floor.

One of the three cases in which the sella turcica was normal in size showed atrophy of the posterior clinoid processes and dorsum sellae. All three patients were further studied by means of pneumencephalography performed through the lumbar route. Obliteration of the anterior portion of the cisterna interpeduncularis together with a concave defect rostrally was demonstrated in one case. (Fig. 1.) Failure to visualize the cisterna chiasmatis and the cephalic half of the cisterna interpeduncularis occurred in another patient. (Fig. 2.) Deformity of the cisterna interpeduncularis by a mass which also projected into the third ventricle was revealed in a third encephalogram. (Fig. 3.) Ventriculography was employed in two cases that presented diagnostic problems. The sella turcica was enlarged and deformed in both but the clinical pictures were most unusual for a pituitary adenoma. The ventriculograms of one of these patients revealed a mass arising from the region of the chiasm which protruded into the cisterna interpedun-

cularis and also elevated the anterior aspect of the third ventricle. On laminagraphic section it was possible to demonstrate a large tumor eroding the anterior wall and floor of the sella and filling the sphenoidal sinus. (Fig. 4.) In the second

TABLE I

Type of Defect	No. of Cases
Bitemporal hemianopsia	63
One eye blind or barely perceiving light; temporal field defect in the other eye	14
Homonymous hemianopsia	6
Bilateral blindness	1
Bilateral light perception	1
Unilateral temporal field defect	2
Light perception in one eye; other eye normal	1
Inferior altitudinal hemianopsia	1
No defect demonstrable	2
Total	91

case, the films showed considerable dilatation of the lateral ventricles and a deformity of the anterior portion of the third ventricle outlining a tumor mass. Concave filling defects in the floors of the frontal horns and an elevation of the cisterna interpeduncularis were also demonstrable. (Fig. 5.) The diagnosis of a chromophobe adenoma was subsequently verified histologically in all five cases studied by pneumencephalography.

Examination of the fundi disclosed optic atrophy in seventy-nine patients. Both

FIG. 3. Case 87. Deformity of cisterna interpeduncularis by a mass also projecting into third ventricle.

discs were atrophic in sixty-two patients while in the other seventeen unilateral funduscopic changes were observed. Papilledema was found in only one case but was reported to have been present in another patient prior to his original operation elsewhere. The various types of visual field defects observed and their incidence is recorded in Table 1.

It was impossible to chart the fields in one case owing to the torpid state of the patient. In another case, the nature of a visual defect which subsequently receded after therapy was not established. Studies of the patients in whom a unilateral tem-
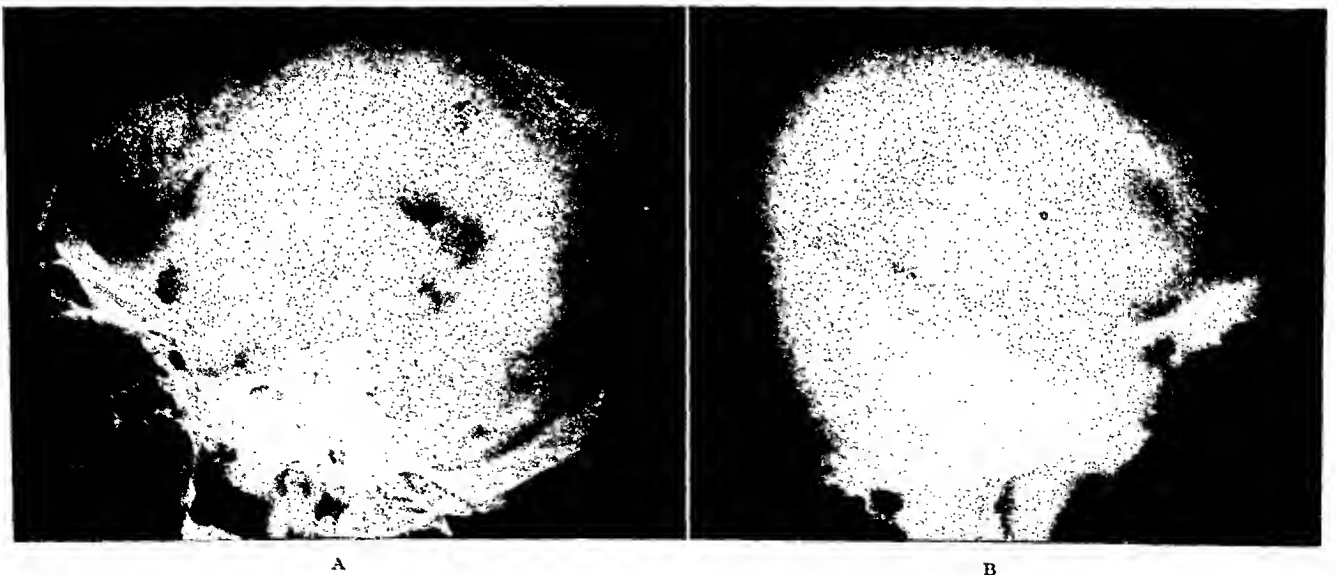
FIG. 4. Case 77. A and B, pneumencephalographic evidence of a mass arising from region of chiasm, projecting into cisterna interpeduncularis and elevating anterior portion of third ventricle is not well demonstrated in the photographs. Advantage of a laminagram is clearly indicated. On the plain films, there is poor visualization of the sella turcica. Laminagram distinctly reveals enlargement of the sella and erosion of the anterior wall and floor.
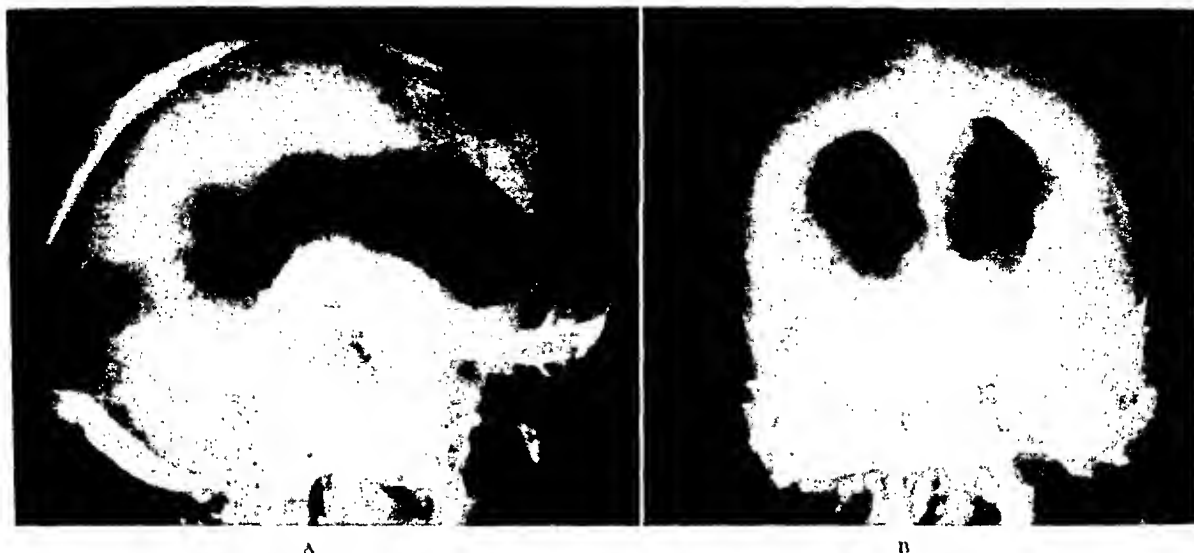
Fig. 5. Case 86. A and b, dilated lateral ventricles and a deformity of the anterior portion of the third ventricle outlining the tumor. There are also concave filling defects in the floors of the frontal horns and an elevation of the cisterna interpeduncularis.

poral field defect was initially discovered revealed a temporal defect in the other eye during the subsequent course of the illness in one and a disappearance of the defect following therapy in the other. On reexamining postoperatively the patient with light perception in one eye and a normal field in the other, an inferior altitudinal hemianopsia was demonstrated. Examination at still a later date revealed further recession of the scotoma and an inferior nasal quadrantic defect remained. The inferior altitudinal hemianopsia which was observed in another patient was most marked in the right field of vision. At operation the tumor was seen to compress the right optic nerve against the optic foramen. This observation, in all probability, accounted for the bizarre visual field.

Extraocular muscle palsies and disturbances referable to the trigeminal nerves were recorded in a few instances. Eight patients complained of diplopia; in four, there were manifest extraocular muscle paralyses. Unilateral ptosis was present in an additional two cases. Trigeminal involvement, both motor and sensory, combined with an ipsilateral ophthalmoplegia was observed in a totally amaurotic patient. Pain in the distribution of the supraorbital nerve was the chief complaint in

another instance though objective evidence of sensory deficit was lacking.

Hemiparesis occurred late in the course of three cases in all of whom the tumor had extended intracranially into the middle fossa. It was associated with Jacksonian seizures in one and with a sensory and motor aphasia in another. Unilateral upper motor neuron signs were elicited in still another patient in whom the tumor had grown into the third ventricle and blocked the foramen of Monro. Convulsions occurred in an advanced case which at necropsy revealed extension of the growth into the third ventricle and the left temporal lobe.

Treatment. It is the practice in this clinic to treat pituitary tumors initially by means of irradiation unless reasons exist which make surgical intervention imperative, chief among which is a severe impairment of vision that makes the danger of amaurosis an imminent one. Most cases in this series were treated in accordance with this view. Careful surveillance of the patient's progress was maintained during radiation therapy, the all important guides being the direction of change of the visual acuity and visual fields. Wherever visual impairment was of a degree to cause concern, treatment was instituted in the

hospital where close cooperation was maintained with the radiologic and ophthalmologic departments. In these cases, visual fields and acuity were examined at least once a day and more often if thought necessary. Provided further loss of vision was halted, even if no improvement was apparent, radiation therapy was continued. If, on the other hand, vision measured in terms of acuity and visual fields deteriorated further, operation was promptly undertaken. Cases favorably affected by irradiation received several such courses of treatment. The technic employed is that described in the monograph of Dyke and Davidoff, the physical factors being: 200 KV constant potential machine, 50 cm. target skin distance, filter of ½ to 1 mm. Cu and 1 mm. Al, and a current of 8 ma. Three portals were used, a right and left temporal, 8 cm. in diameter, and a central forehead area, 5 to 6 cm. in diameter. Treatment was administered daily or every other day and consisted of 200 r to one portal per day, rotating the several portals in sequence. The total amount of radiation per portal varied between 800 r and 1000 r, so that a complete course consisted of 2,400 to 3,000 r measured in air without backscattering. Usually three or four such courses of treatment were prescribed, spaced at intervals of four to eight weeks, depending on the degree of visual disturbance. Inasmuch as the patients in this series were not treated by any one radiologist, the plan of therapy just outlined was not rigorously adhered to in every case. In general, however, it is a schematic representation of the treatment administered to most patients subjected to radiation in our series.

There were three indications for operation: (1) an advanced degree of visual loss bordering on amaurosis; (2) progressive visual impairment despite adequate irradiation or an arrest of visual failure at an unsatisfactory level; (3) a clinical picture not altogether typical of a pituitary tumor and compatible with another type of chiasmal lesion.

Operation was performed in all cases through a transfrontal approach. A coronal skin incision has in recent years largely replaced the Cushing and Frazier types of flap except in the case of male patients who are bald or whose hairline is receding. It is

TABLE II

RADIATION THERAPY WITH IMPROVEMENT

Cases	Degree of Improvement	Duration of Follow-up
1. F. Y. (M. H. #42294)	Slight	5 mo.
2. B. L. (J. H. #251856)*	Marked	5 yr.
3. M. S. (M. H. #251856)*	Marked	11 mo.
4. H. R. (J. H. #41698)	Marked	6 yr.
5. P. J. (non-hospitalized case)	Slight	10 yr.
6. M. K. (non-hospitalized case)	Slight	8 yr.
7. A. C. (non-hospitalized case)	Marked	2 yr.
8. A. M. (non-hospitalized case)	Marked	2½ yr.
9. E. S. (non-hospitalized case)	Marked	1½ yr.
10. B. S. (non-hospitalized case)	Marked	5½ yr.
11. H. C. (non-hospitalized case)	Marked	7½ yr.
12. J. B. (non-hospitalized case)	Marked	1 yr.
13. R. H. (non-hospitalized case)	Marked	4 yr.
14. A. F. (non-hospitalized case)	Marked	11 mo.
15. M. B. (non-hospitalized case)	Marked	17 mo.
16. B. B. (non-hospitalized case)†	Marked	3 yr.
17. E. B. (non-hospitalized case)‡	Marked	1½ yr.

* Return of menses; had baby.
† Relief of headaches; normal visual acuity and visual fields prior to institution of therapy.
‡ Relief of headache; retention of good vision existing prior to therapy.

customary in this clinic to operate on the side of the non-dominant hemisphere regardless of whether or not this happens to be the side of the more seriously affected eye. The chiasmal region is reached through an intradural approach, reserving the extradural route, in which the dura is opened along the sphenoid ridge, for cases undergoing secondary operations.

In ninety of the ninety-three cases, adequate data exist to permit analysis of the results of treatment. These are listed in Tables II through VIII.

Fifty-nine patients were treated initially with irradiation. Improvement resulted in seventeen patients, 29 per cent. (Table II.) In another seven cases, 12 per cent, there was improvement with subsequent regres-

TABLE III
RADIATION THERAPY—IMPROVEMENT WITH SUBSEQUENT RECURRENCE

Cases	Duration of Improvement	Subsequent Treatment	Subsequent Course
18. B. B. (J. H. #263335)	Marked improvement; regression after 3½ yr.	Further x-ray therapy	Receiving treatment
19. I. S. (J. H. #263103)	Marked improvement; regression after 6 mo.	Further x-ray therapy failed to halt decline of vision; operation	Marked improvement; 3½ yr. follow-up
20. A. C. (J. H. #260361)	Slight improvement; regression after 3 yr.	Operation	Postoperative death
21. L. W. (non-hospitalized case)	Marked improvement; regression after 5 yr.	Further x-ray therapy	Improvement; 7 yr. follow-up
22. M. B. (J. H. #254458)	Slight improvement; regression after 20 yr. (rapidly became amaurotic)	Further x-ray therapy	Relief of headache; no return of vision; died 2 yr. later; details unknown
23. B. K. (N. I. #30134) Mixed tumor	Improvement; regression after 4 yr.	Further x-ray therapy; progressive mental symptoms; operation 4 yr. later	Died 3 mo. after operation
24. R. H. (J. H. #285068) Cystic adenoma	Slight improvement 8 mo., recurrence of headache	Operation	Marked improvement; recurrence headache and visual loss 4 yr. later; marked improvement with x-ray therapy; recurrence 3 yr. later; transient improvement with x-ray therapy; re-operated; slight improvement, 1½ yr follow-up since second operation

TABLE IV
RADIATION THERAPY—UNIMPROVED—VISION NO WORSE

Case	Follow-up	Additional Data
25. I. F. (non-hospitalized case)	17 mo.	
26. D. S. (non-hospitalized case)	3 yr.	
27. E. D. (non-hospitalized case) mixed tumor clinically	16 yr.	
28. P. D. (non-hospitalized case)	3 yr.	
29. A. S. (non-hospitalized case)	10 mo.	
30. C. M. (non-hospitalized case)	7 mo.	
31. M. H. (M. H. #41268)	9 mo.	
32. A. L. (J. H. #255333)	......	Operated upon following trial of x-ray therapy; marked improvement; 5 yr. follow-up
33. I. S. (J. H. #247534)	......	Operated upon following trial of x-ray therapy; slight improvement; 1½ yr. follow-up; died of cerebral hemorrhage
34. H. S. (N. I. #10515-H) Cystic adenoma	......	Operated upon; postoperative death

TABLE V

RADIATION THERAPY—FURTHER VISUAL LOSS

Case	Subsequent Treatment	Subsequent Course
35. H. K. (N. I. #16515-H)	Operation; postoperative x-ray therapy	Slight improvement; 13 yr. follow-up
36. L. G. (J. H. #233016)	Operation	Slight improvement; 7 yr. follow-up
37. E. J. (J. H. #265836)	Operation	Marked improvement; 4 yr. follow-up
38. N. G. (J. H. #246192)	Operation	Slight improvement; 2 yr. follow-up
39. B. F. (J. H. #268184)*	Operation; postoperative x-ray therapy	Marked improvement; 4 yr. follow-up
40. A. W. (J. H. #241904) Cystic adenoma	Operation	Marked improvement; 6 yr. follow-up
41. I. W. (M. H. #39760) Mixed tumor	Operation	Slight improvement; 20 mo. follow-up
42. E. G. (J. H. #259616) Cystic adenoma	Operation	Slight improvement; 4½ yr. follow-up
43. C. S. (J. H. #221701) Cystic adenoma	Operation; postoperative x-ray therapy	Marked improvement; 8 yr. follow-up
44. P. C. (J. H. #260198)	Operation; postoperative x-ray therapy	Marked improvement; 3 yr. follow-up
45. P. O. (M. H. #41288)	Operation	Slight improvement; 5 mo. follow-up
46. M. O. (M. H. #39803)	Operation	Marked improvement; 1½ yr. follow-up
47. E. B. (non-hospitalized case)	Refused operation	
48. H. R. (non-hospitalized case (Mixed tumor clinically)	Operation elsewhere	
49. C. G. (non-hospitalized case)	Refused operation	
50. R. F. (J. H. #261704)	Operation	Loss of vision one eye; loss of temporal field in other eye with maintenance visual acuity 20/40; 5 yr. follow-up
51. A. S. (N. I. #21537)	Operation	Postoperative clot; vision much worse after operation; unimproved; 4 yr. follow-up
52. R. M. (J. H. #253448)	Operation	Slight improvement postoperatively; died 10 yr. later of recurrence
53. B. A. (N. I. #27341-H)	Operation	Postoperative death
54. S. S. (J. H. #261926)	Operation	Postoperative death
55. Y. K. (J. H. #237623)	Operation	Prepared for operation; failure of respiration during induction of anesthesia
56. B. S. (J. H. #270951)† (M. H. 40549)	Operation	Marked improvement; recurrence 3 yr. later; no improvement with x-ray therapy; reoperated; postoperative death; Marked improvement; recurrence within 2 yr.; x-ray therapy without improvement; reoperated; improved; beginning regression again within a year following operation

* Return of menses; had baby.
† Return of menses following first operation; had baby.

TABLE V (Continued)

Case	Subsequent Treatment	Subsequent Course
57. A. G. (N. I. #12295-H).............................	Operation	Improved markedly; recurrence 4 yr. later; reoperated; unimproved for 5 yr.; further decline of vision; third operation; postoperative x-ray therapy; amaurosis; death 3 mo. following third operation
58. C. H. (J. H. #283060) Cystic adenoma.............·......	Operation	Marked improvement; 2 yr. follow-up

TABLE VI

RADIATION THERAPY—PRE- AND POSTOPERATIVELY—VARYING EFFECTS

Case	Course
59. I. S. (J. H. #264399)................	Essentially unimproved following one course x-ray therapy; failure of vision following operation and postoperative x-ray treatment; fourth course of radiation therapy with improvement in one eye and transient improvement in other eye; still another course of radiation therapy with continued improvement in one eye and delayed improvement in other eye

TABLE VII

OPERATION COMBINED WITH POSTOPERATIVE IRRADIATION—RESULTS

Case	Degree of Improvement
60. S. S. (N. I. #12416-H).............:..	Improvement of vision in one eye; other eye remained amaurotic; 4 yr. follow-up
61. M. R. (B. I. #91771)................	Marked improvement; 10 yr. follow-up
62. M. K. (N. I. #32889)................	Marked improvement; 9 yr. follow-up
63. A. B. (J. H. #222720)................	Marked improvement; 8 yr. follow-up
64. H. S. (N. I. #23257-H).......:......	Marked improvement; 4 yr. follow-up
65. J. M. (J. H. #215008) Mixed tumor...	Marked improvement; 9 yr. follow-up
66. L. S. (J. H. #246861)................	Marked improvement; 6 yr. follow-up
67. M. C. (M. H. #41746)................	Marked improvement; 6 mo. follow-up
68. A. T. (J. H. #272125)................	Unimproved; 2 yr. follow-up; (severe visual impairment preoperatively)
69. M. T. (N. I. #22488)................	Unimproved; 12½ yr. follow-up; (severe visual impairment preoperatively)
70. R. B. (B. I. #84165) Cystic adenoma..	Unimproved; 6 mo. follow-up; (severe visual impairment preoperatively)
71. M. K. (J. H. #244685)	Unimproved postoperatively; (severe visual impairment preoperatively); died 10 mo. later as a result of injury
72. L. F. (J. H. #241925)* Mixed tumor...	Marked improvement; beginning regression of vision in one eye 6 yr. after operation
73. I. W. (J. H. #208671)................	Progressive failure of vision; refused reoperation; received 53,100 r over a 4-yr. period; died 8 yr. postoperatively
74. O. G. (N. I. #14191-H)................	Slow progressive visual loss; 15 yr. follow-up
75. T. C. (J. H. #280772)................	Slight improvement; recurrence after 6 yr., failure of x-ray therapy to halt visual loss; reoperated upon; death 6 wk. later
76. L. H. (J. H. #222976) Mixed tumor....	Slight improvement; recurrence within 4 mo.; reoperation plus x-ray therapy; continued to fail; died 10 mo. later following an operation elsewhere

* Became a father.

sion. One of these patients improved a second time with further radiation therapy. (Case 21, Table III.) Visual failure was halted in an additional 10 patients, 17 per cent, although no improvement in vision occurred. (Table IV.) Nevertheless, since

after a fourth course of irradiation was improvement apparent; in one eye, improvement was of transient duration. A fifth course of radiation therapy was administered; and while improvement in the poor eye did finally occur, it was long

TABLE VIII
RESULTS FOLLOWING OPERATION ALONE

Case	Course
77. L. B. (J. H. #285402)................	Marked improvement; 2 yr. follow-up
78. H. S. (non-hospitalized case) transfrontal operation 1927 by Dr. Cushing.....	Unimproved; useful vision one eye; 11 yr. follow-up
79. A. B. (non-hospitalized case)* Transfrontal operation 1931 by Dr. Cushing.	Unimproved; 13 yr. follow-up
80. A. W. (M. H. #40077) Transphenoidal operation 1927 by Dr. Cushing.	Maintained vision in right eye; left eye blind; recurrence after 17 yr.; x-ray therapy failed to halt visual loss; operation; postoperative death
81. M. R. (J. H. #279166) Transfrontal operation 1932 by Dr. Cushing Mixed tumor.	Marked improvement; recurrence 12 yr. after operation; irradiation failed to halt visual decline; reoperated; died 1 mo. postoperatively.
82. E. S. (N. I. #33280) Transfrontal operation 1935 by Dr. Frazier.	Recurrence 2 yr. later; reoperated 1937; continued to fail; expired 1941
83. V. D. (M. H. #22174)† Transphenoidal operation 1922 by Dr. Cushing.	Improvement; recurrence in 1927; improvement with x-ray therapy; recurrence in 1930; further irradiation in 1933 without improvement; transfrontal operation with postoperative irradiation in 1933; marked improvement maintained since then
84. I. D. (V. C. #289850) Transphenoidal operation 1911 by Dr. Cushing.	Improved considerably; recurrence in 2 yr.; subtemporal decompression and reoperation through transphenoidal route by Dr. Cushing; improved; recurrence 5 yr. later; almost completely amaurotic in 1931
85. J. Y. (M. H. #41444) Transphenoidal operation 1927 by Dr. Cushing.	Recurrence in 1934; transfrontal operation and postoperative irradiation; improved; recurrence within a yr.; x-ray therapy without improvement; relatively little change until 1946; further regression with mental changes and convulsions; third operation with improvement of mental condition; vision no better
86. J. S. (J. H. #255405)................	Postoperative death
87. R. C. (J. H. #210323)................	Postoperative death
88. N. A. (N. I. #28059-H) Mixed tumor..	Postoperative death
89. A. C. (N. I. #13507-H) Mixed tumor..	Postoperative death
90. L. K. (J. H. #217843)................	Improved; recurrence after 22 mo.; x-ray therapy; marked improvement; 5 yr. follow-up

* Return of menses; had baby.
† Became a father in 1928.

operation was deemed advisable and performed in three of these ten cases, from a practical standpoint, x-ray therapy was efficacious in only seven of them, 12 per cent. Further visual failure despite irradiation occurred in twenty-four cases, 41 per cent. (Table V.) Of twenty-three patients worse or unimproved following radiation therapy and subjected to operation, cystic adenomas were found in only five. The case listed in Table VI was unique in several respects, including the response to roentgen treatment. (Fig. 14.) Preoperative radiation effected little change and following operation vision deteriorated further. Only

delayed. Case 90 included in Table VIII also merits comment. This patient was originally operated upon because a suprasellar meningioma was suspected. What appeared to be a complete removal of the tumor was accomplished and the tissue was reported to be a meningioma. Recurrent symptoms twenty-two months later led to a review of the sections of the tumor at which time the correct diagnosis of a chromophobe adenoma was established. Radiation therapy following symptoms of recurrence resulted in marked improvement which has been maintained for a five-year period.

The results achieved with radiation therapy in this series are shown in Table IX.

In summary then, thirty-five patients (59 + per cent) showed a favorable response to irradiation, varying in degree

TABLE IX

RESULTS FOLLOWING RADIATION THERAPY

1. Improved {slightly............... 3 } 17
            {markedly............... 14}
2. Improvement with subsequent regression....... 7
3. Unimproved but no worse................... 10
   (Three were subsequently operated upon)
4. Varying response on different occasions with
   eventual improvement.................... 1
   (Case 59)
5. Further visual failure..................... 24
                                             ——
   Total................................... 59

from an arrest of progressive visual failure to mark improvement of visual acuity and visual fields.

A total of twenty-eight patients were operated upon following radiation therapy. This included some who were unimproved, those in whom vision deteriorated still further and several who manifested evidence of recurrence following a favorable response to radiation. Additional postoperative radiation was administered in four of these patients. The results are listed in Table X.

TABLE X

RESULTS OF OPERATION FOLLOWING RADIATION THERAPY

1. Improved {slightly............... 8 } 21
            {markedly............... 13}
2. Progressive failure of vision................... 1
   (Case 59)
3. Vision worse following operation.............. 1
   (postoperative clot)
4. Postoperative death....................... 4
5. Death 4 mo. after operation (pituitary failure).. 1
6. Subsequent recurrence...................... 5

There were five patients with recurrence following operation. In four of these, recurrence appeared within four years and it was subsequently necessary to reoperate upon all of them. Concerning the fifth case, detailed information was not available. However, he is known to have expired ten years after operation with evidence of recurrence. Regression in one patient (Case 24) was favorably affected by roentgen treatment for a period of four years. Of the four patients for whom a second operation

was performed, one has remained slightly improved; another is showing signs of further recurrence; a third was unimproved and eventually required still another operation which failed to prevent further loss of

TABLE XI

RESULTS OF OPERATION COMBINED WITH POSTOPERATIVE RADIATION THERAPY

Improved {slightly............... 2 } 10
         {markedly............... 8 }
Improved markedly in one eye; remained blind in
   other eye................................. 1
Unimproved................................. 4
(Vision severely impaired in all 4 cases preoperatively)
Progressive failure of vision.................... 2
Recurrences................................. 3

vision and the patient expired three months later; one died postoperatively.

In seventeen cases treatment consisted of operation followed by irradiation. (Table VII.) The results obtained are shown in Table XI.

Of the two patients who continued to lose vision, one has done so slowly over a fifteen-year postoperative period. Vision in the second patient deteriorated more rapidly and the patient expired eight years after operation. Reoperation having been refused, an enormous amount of irradiation, 53,100 r over a four-year period, was administered but to no avail. Recurrences occurred in three cases. In one evidence of slight regression occurred recently; and should this be confirmed, in all probability a course of radiation therapy will be recommended. Recurrence in a second case was treated at first with irradiation and when this proved unsuccessful another operation was performed. Death occurred six weeks later. The third patient who suffered from a recurrence was reoperated upon and given roentgen therapy postoperatively but prevention of visual failure was not achieved. He expired following a third operative procedure performed elsewhere.

The cases listed in Table VIII, which records the results following operation alone, do not lend themselves to analysis as a whole. Eight of them had previously been operated upon elsewhere and of these six were seen in this clinic because of recurrent

symptoms. There was considerable variation in their course so that no generalizations can be drawn. Four patients in this group (Cases 77, 86, 87, 90) were operated upon because the diagnosis of pituitary adenoma was not clear. Three showed atypical clinical features which introduced some question as to diagnosis; the fourth was thought to harbor a suprasellar meningioma. Two additional cases, both postoperative fatalities, are included although had they survived, they would undoubtedly have received postoperative radiation therapy.

Exclusive of recurrences, operation was performed in fifty-two cases of the entire series of chromophobe adenomas. There were three postoperative clots all of which were recognized and evacuated. Eight postoperative deaths occurred with a resultant mortality figure of 15.4 per cent. In one of these cases, the tumor could not be visualized at operation by virtue of its location (Case 87, see case report). In another, the tumor had extended into the third ventricle, necessitating a surgical approach through the corpus callosum (Case 86, see case report). Cerebral thrombosis was the cause of death in a third case. A fourth patient was in a semi-comatose state prior to operation. Still another had an extensive intracranial extension along the base of the skull toward the foramen magnum into the posterior fossa. Three patients expired presumably because of postoperative cerebral edema although no permission for postmortem examination was obtained. Reoperation because of recurrence was performed in nine cases. There were two postoperative fatalities. Two patients required a third operation. Both survived the surgical procedure though one expired three months later.

CASE REPORTS

Improvement Following Irradiation. B. L. (Case 2). Jewish Hospital, No. 251856. A thirty-five year old white female was admitted on March 3, 1942, because of headache and progressive loss of vision of four months' dura-

tion. She was seven months pregnant. Limitation of the peripheral fields of vision was first noted while driving an automobile.

On examination a bitemporal hemianopsia was demonstrable. Visual acuity on the left was 20/40, on the right questionably 2/200. Roentgen examination of the skull revealed enlargement of the sella turcica with considerable thinning of the posterior clinoids, dorsum sellae and floor. Considering the degree of visual impairment, immediate operation ordinarily would have been the procedure of choice. In view of the fact, however, that the patient had no other children, was thirty-five years old and extremely eager to continue with her already advanced pregnancy, a carefully supervised trial of radiation therapy was instituted. Irradiation was administered in daily doses of 200 r. Three portals, two lateral and one anterior, were treated in rotation, a total of 800 r being given to each field over a period of twelve days. Her visual fields were examined every day. Within a week's time, a distinct improvement in visual acuity and peripheral fields was observed. At the time of discharge from the hospital on March 17, 1942, visual acuity on the left was 20/20 and on the right, 20/100. On April 16, 1942, a second course of radiation therapy was begun and a total of 3,000 r administered. She continued to maintain visual improvement. On May 28, 1942, a normal baby was delivered by cesarean section. Following the birth of her child, a third course of irradiation was prescribed. In June, 1942, visual acuity was 20/20 on the left and 20/40 on the right. The peripheral fields continued to expand. In June, 1943, however, there was some recurrence of headache and a slight shrinkage of the peripheral fields. Another course of roentgen therapy was administered, totalling 3,000 r. Improvement promptly ensued. In September, 1943, menses were resumed. The patient was examined again on March 14, 1946, at which time the right optic disc showed a slight degree of atrophy. Her condition otherwise was very satisfactory. Perimetric studies in July, 1947, revealed full peripheral fields and visual acuity of 20/20 on the left and 20/20 −2 on the right (Fig. 6.)

B. S., (Case 10), non-hospitalized patient. A twenty-seven year old white female was seen on January 29, 1942, because of visual impairment. She had consulted an ophthalmologist in February, 1941, who found a bitemporal hemi-

anopsia and visual acuity of 20/20 on the right and 20/30 on the left. An enlarged sella turcica was also discovered. It was further learned that her menses had begun at the age of eleven but had ceased when she was about fifteen years old. On a few occasions there was some slight

June 2, 1941, 1,500 r having been administered to each of three portals. The patient stated her eyesight now was worse than at the time treatment was completed.

Examination revealed a short, obese, pale young woman with a thin, dry skin, tapering
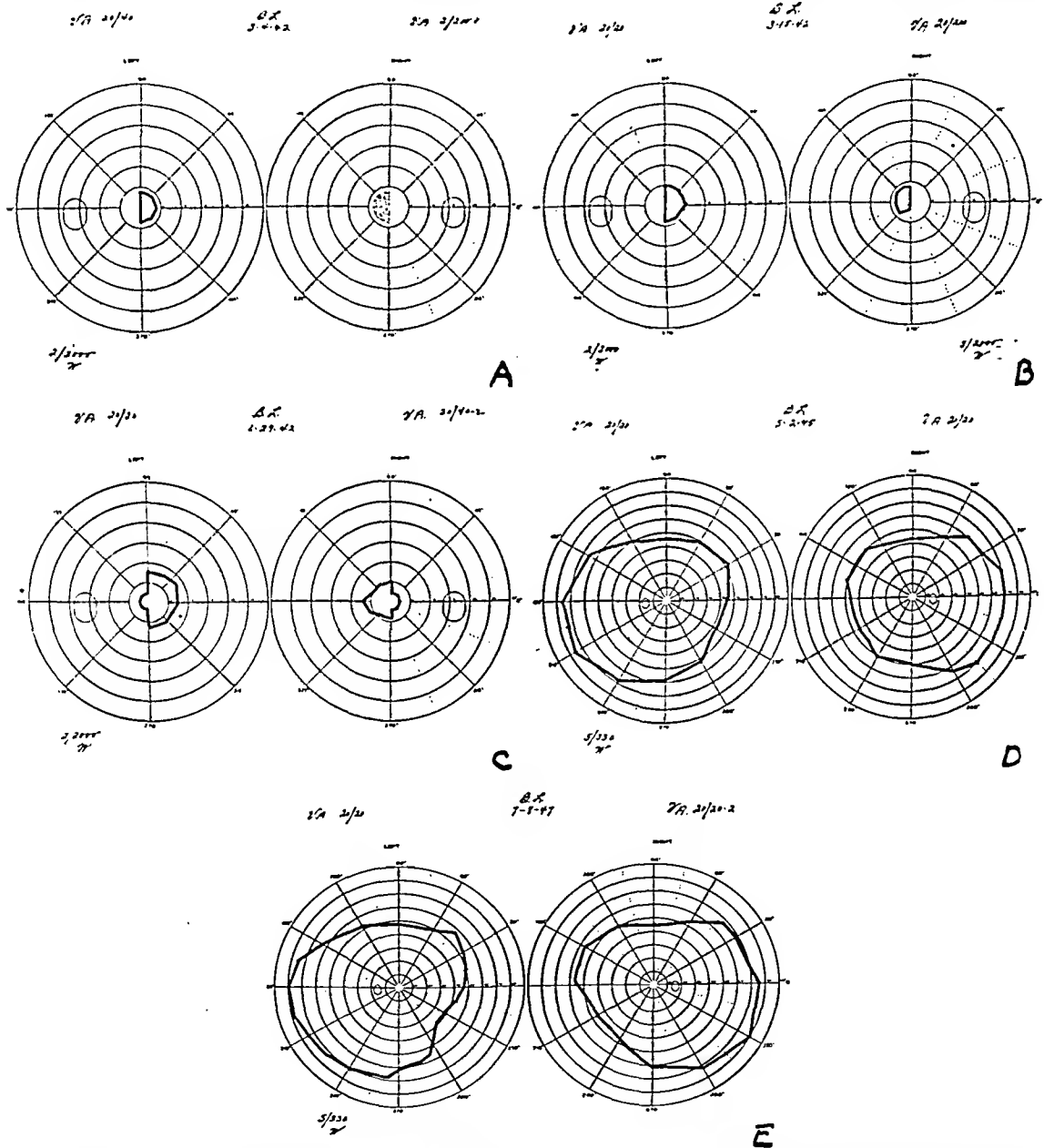
FIG. 6. Case 2. A, central fields prior to irradiation; B, central fields eleven days after instituting treatment; C, central fields following third course of therapy; D, peripheral fields, three years follow-up; E, peripheral fields, five years follow-up.

staining brought about by the injection of female sex hormone. She had gained about 10 or 12 pounds during the past six to seven years. Radiation therapy was given from April 11 to

fingers and fine, silky hair on her head. There was no axillary hair whatever, only a small amount of hair over the labia and practically none over the pubis. The external genitali were

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infantile and the breasts underdeveloped. There was pallor of the optic nerve heads. An x-ray of her skull showed an enlarged sella turcica with a thinning of the posterior clinoids and dorsum sellae. Visual field studies revealed a bitemporal hemianopsia; the visual acuity

A course of radiation therapy was started on February 19, 1946, and completed on April 19, 1946. A total of 3,390 r was applied to three fields. The patient improved subjectively but in August, 1946, further diminution occurred in the visual fields and in the acuity of the left

V.A. $\frac{20}{50}$ - 1   B.S.   V.A. $\frac{20}{25}$ A   B
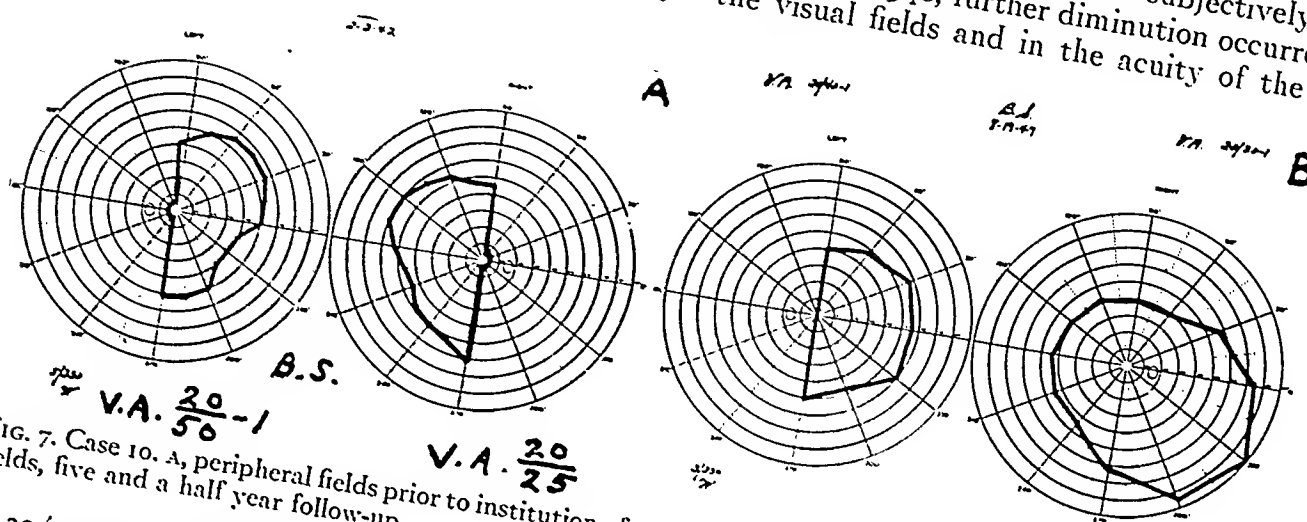
FIG. 7. Case 10. A, peripheral fields prior to institution of second course of irradiation; B, most recent peripheral fields, five and a half year follow-up.

was 20/25 on the right and 20/50 — 1 on the left. A further trial of radiation therapy was deemed advisable and another course administered from February 2 to March 20, 1942. Again 1,500 r were applied to each of three portals. Visual acuity improved and the right temporal field began to expand. There has been little change since 1944. Her visual acuity at present is 20/20 — 1 on the right and 20/40 — 1 on the left. (Fig. 7.) She complains of no headache and in general, feels quite well. The amenorrhea persists.

E. B., (Case 17), non-hospitalized patient. A thirty-nine year old white female was seen on February 18, 1946, because of blurred vision. She was well until three years ago at which time her menses ceased abruptly. She received injections of gonadotropic hormones without any effect on the amenorrhea. A few months previously vision became blurred. She consulted an ophthalmologist who discovered a bitemporal hemianopsia. Headache was never a symptom. There was no disturbance of libido. Neurologic examination was negative except for the visual field changes. Visual acuity was 15/30 on the right and 15/70 on the left. Funduscopic examination revealed no change in the optic discs. Skull films indicated marked enlargement of the sella turcica with atrophy of the posterior clinoids, sellar floor and dorsum sellae.

eye. She was seen again in this clinic in October and further irradiation recommended. From November 4, 1946, to December 20, 1946, she received a total of 3,300 r. Vision improved gradually over a period of several months. When last seen on September 9, 1947, she was asymptomatic. Visual acuity on the right was 15/20 and on the left, 15/50. The visual fields had improved a good deal. (Fig. 8.)

M. S., (Case 3), Montefiore Hospital, No. 41698. A fifty-two year old white male came under observation in October, 1946, because of visual failure of about one year's duration. Impairment of sight was more pronounced in the right eye. For about a similar length of time, he had experienced intermittent headache, frequently quite severe in intensity. Libido and potency had been diminished for several years, and in the past few months he had been completely impotent. Operation was advised at another hospital but refused by the family. Examination revealed a pale, somewhat obese patient with a thin skin, sparse beard and tapering fingers. There was slight bilateral optic atrophy. Visual field studies revealed a left homonymous hemianopsia. Visual acuity was 7/200 on the right and 18/200 on the left. Roentgenograms of the skull showed enlargement of the sella turcica with atrophy of the posterior clinoids, dorsum sellae and sellar floor. Destructive changes were also observed

in the left sphenoid ridge, left fronto-orbital region, left petrous apex and possibly also the right petrous apex. The extensive bony destructive changes raised some question as to the diagnosis, but the composite clinical picture was clearly that of a chromophobe pituitary adenoma.

F. Y., (Case 1), Montefiore Hospital, No. 42294. A forty-nine year old white female was admitted to the hospital on March 19, 1947, because of visual impairment. Eight years ago dimness of vision was first observed. During the year prior to admission, it became increasingly difficult for her to read. She complained of no
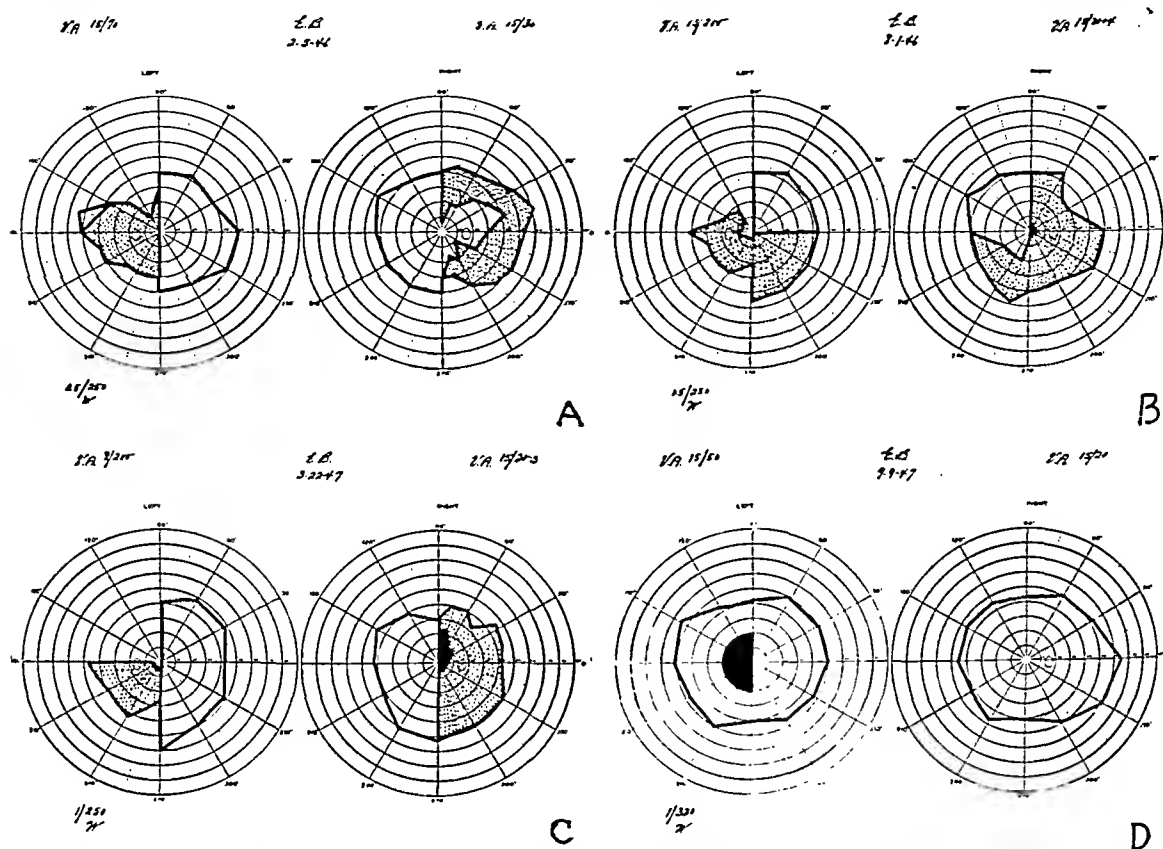
FIG. 8. Case 17. A, peripheral fields before therapy; B, peripheral fields, three months after first course of irradiation; C, peripheral fields, three months after second course of therapy; D, most recent fields, six months later.

Radiation therapy was instituted and in October and November, 1946, he received 2,925 r. There was some improvement in visual acuity on the left but headache was worse. Surgical intervention was again proposed and the patient scheduled for operation. During the induction of anesthesia, however, his condition became precarious and the procedure was cancelled. Further irradiation was, therefore, prescribed and two subsequent courses, each of 3,000 r, were administered between December 27, 1946, and January 31, 1947, and between March 10, 1947, and April 11, 1947. Visual acuity and fields showed remarkable improvement. On September 18, 1947, his vision was 15/20 in both eyes and the peripheral fields had expanded considerably. (Fig. 9.) He was completely relieved of headache.

headache. Menses were regular though there had been a period of amenorrhea between the ages of thirty-eight and forty-two.

Neurologic examination revealed pallor of both optic discs and a bitemporal hemianopsia. Visual acuity in the right eye measured 15/100 − 1 and 15/200 in the left eye. Roentgenograms of the skull showed considerable enlargement and atrophy of the sella turcica.

Radiation therapy was instituted and between March 25, 1947, and April 29, 1947, a total dose of 2,900 r was administered. There was slight but definite improvement in vision. On April 3, 1947, visual acuity in the right eye was 15/70 + 2 and the visual fields had expanded slightly. On August 26, 1947, visual acuity on the right measured 20/40 and on the left 20/100. There was further improvement in

the peripheral fields. (Fig. 10.) A second course of irradiation has been started.

Further Visual Failure Following Irradiation Therapy. N. G., (Case 38), Jewish Hospital, No. 246192. A twenty-eight year old white male

visual field studies were continued. He was discharged from the hospital on August 28, 1941.

Visual failure was progressive despite irradiation and after 4,500 r had been administered he was readmitted on October 5, 1941. Visual
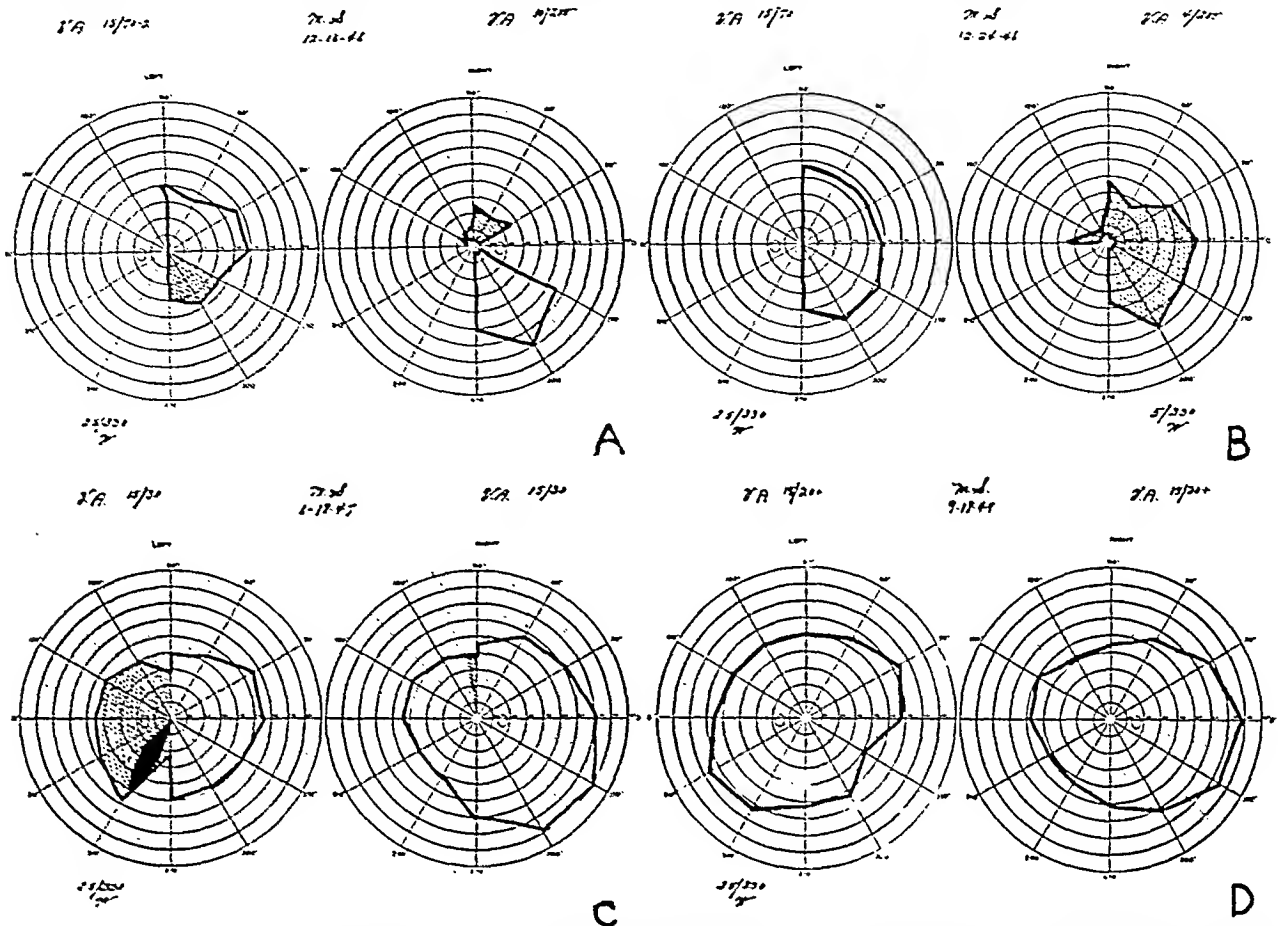
FIG. 9. Case 3. A, peripheral fields, one month following first course of irradiation; B, peripheral fields prior to second course of treatment; C, same, two months after third course of therapy; D, most recent peripheral fields, five months after completion of irradiation.

was first admitted to the hospital on August 21, 1941, because of headache of a little over one year's duration. For two months prior to admission the severity of the headache had increased markedly and was accompanied by visual impairment. He particularly noted that while playing ball he was unable to see the ball from various positions. There was also dimness of vision in the left eye.

On examination, pallor of both optic discs was noted. Visual field studies revealed a bitemporal hemianopsia, more marked on the left. Visual acuity was 20/30 − 2 on the right and 20/70 − 2 on the left. Skull roentgenograms showed a marked ballooning of the sella turcica with loss of density of the sellar floor and posterior displacement of the dorsum sellae. Radiation therapy was begun on August 26, 1941, and while this treatment was being given,

acuity was 20/30 + 2 on the right and 5/200 on the left. A right transfrontal craniotomy was performed on October 6, 1941, and an intracapsular partial removal of a pituitary tumor accomplished. Microscopic examination of the tumor revealed it to be a chromophobe adenoma. Postoperatively he received a course of irradiation totalling 3,000 r which was completed in November, 1941. Headache was relieved and he felt much improved. Libido and potency were diminished but present. The right central field of vision improved considerably and visual acuity improved to 20/20. There was no change of visual acuity on the left and no restitution of the temporal field on that side. When last seen in October, 1943, his condition and visual status were the same. (Fig. 11.)
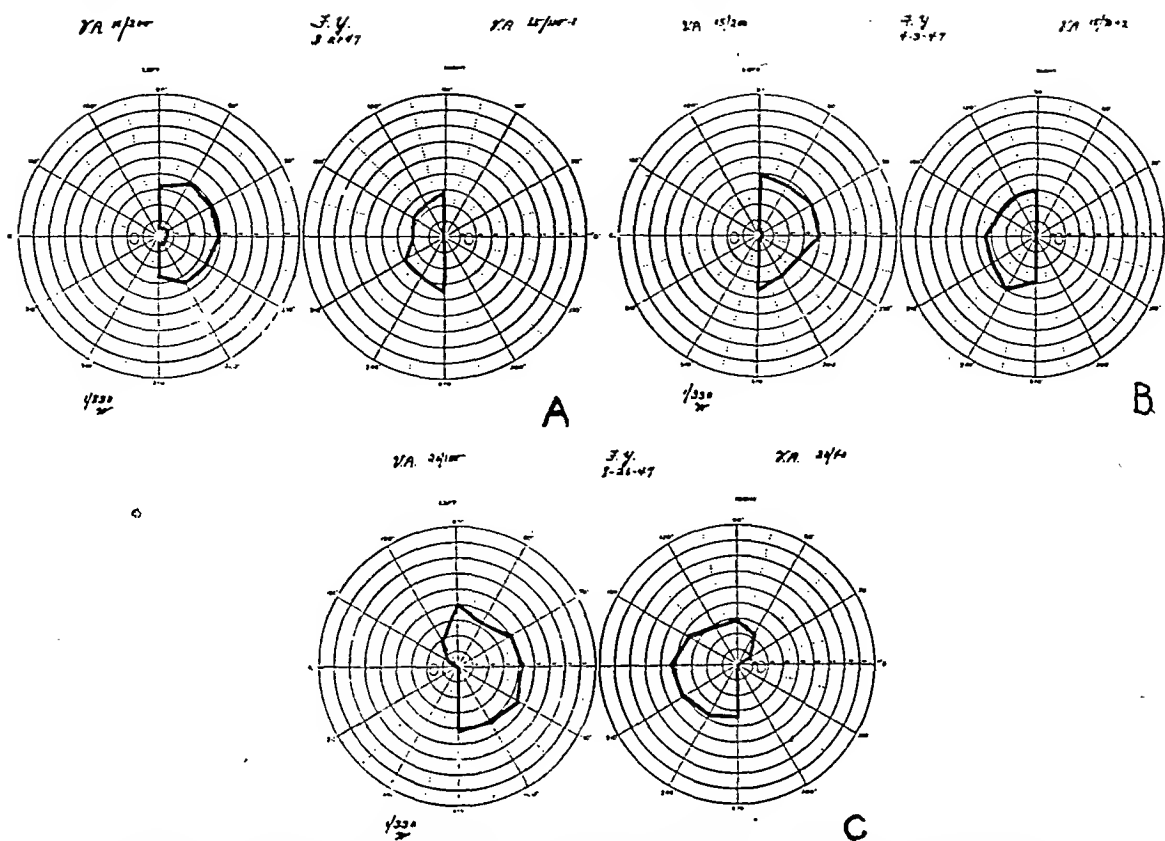
FIG. 10. Case 1. A, peripheral fields before treatment was started; B, peripheral fields after treatment was begun; C, peripheral fields, four months, after completion of therapy.
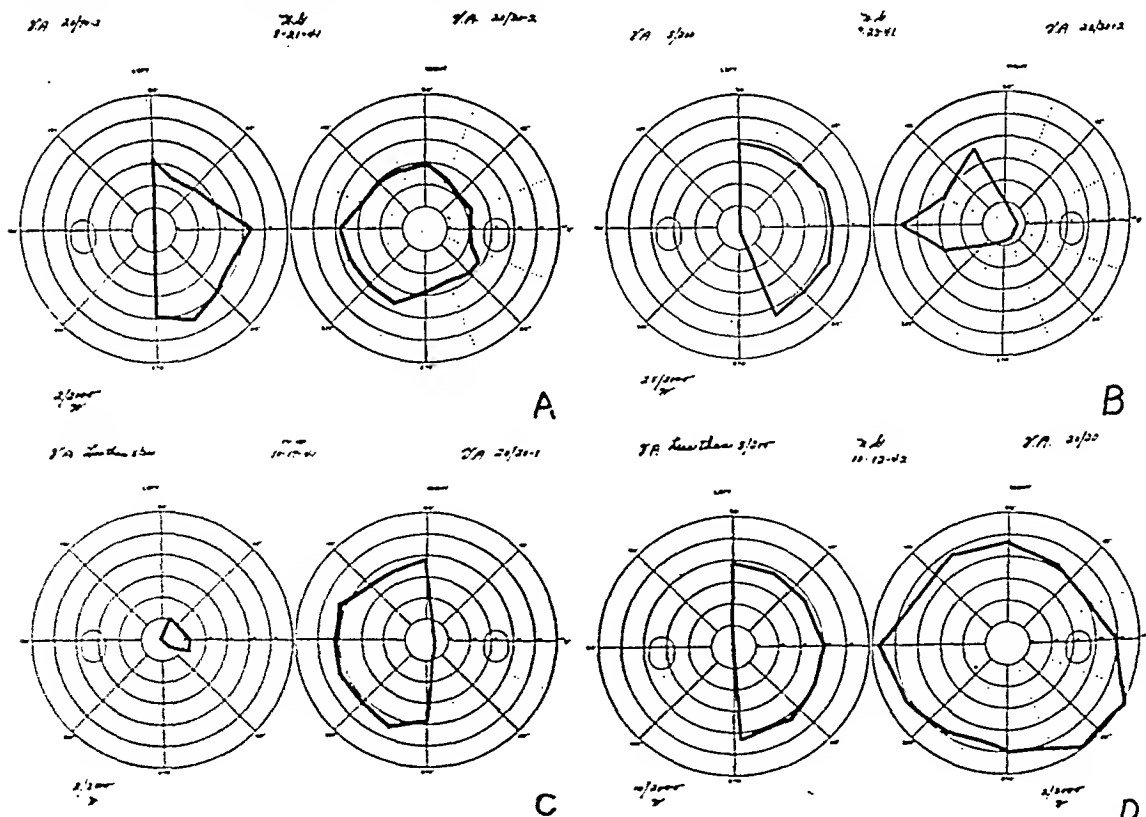
FIG. 11. Case 38. A, central fields prior to irradiation; B, central fields after institution of treatment; C, same after operation; D, central fields one year after operation and postoperative irradiation.

Improvement Following Operation and Postoperative Irradiation. M. C., (Case 67), Montefiore Hospital, No. 41746. A thirty-eight year old white male was admitted on January 28, 1947, because of blurring of vision in the left eye of about one year's duration. This was his

and a limited amount of body hair. He was married and had three children.

Examination revealed a well developed but pale man with a completely negative neurologic status except for the visual field studies. These revealed a bitemporal hemianopsia, more ad-
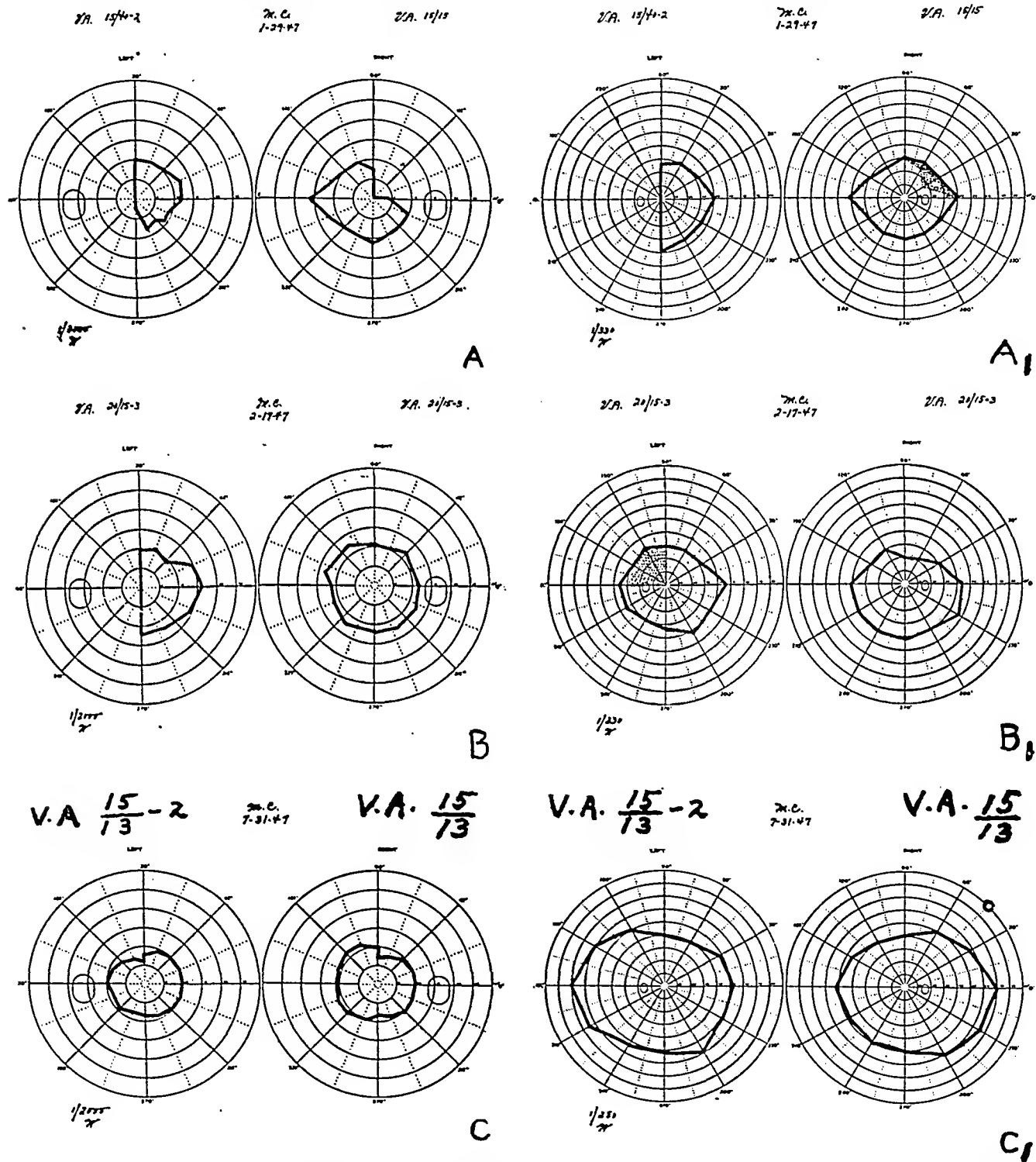
FIG. 12. Case 67. A-A₁, central and peripheral fields prior to operation; B-B₁, postoperative central and peripheral fields; C-C₁, same four, months after completion of irradiation.

only complaint. He denied any change in his body configuration or alteration in libido or potency. He always had a rather sparse beard

vanced in the left eye. Visual acuity was 15/15 in the right eye and 15/40 − 2 in the left eye. Roentgenograms of the skull showed a thick-

ened vault, and *a sella turcica of normal size,* but with atrophy of the posterior clinoids and dorsum sellae. Pneumencephalography was performed revealing a chiasmal mass continuous with the sellar contents, obliterating the cisterna chiasmatis and the anterior half of the

L. S., (Case 66), Jewish Hospital, No. 246861. A fifty-four year old female was admitted on September 10, 1941, because of headache of four years' duration and blurring of vision of four week's duration. Impairment of sight had become rapidly worse.
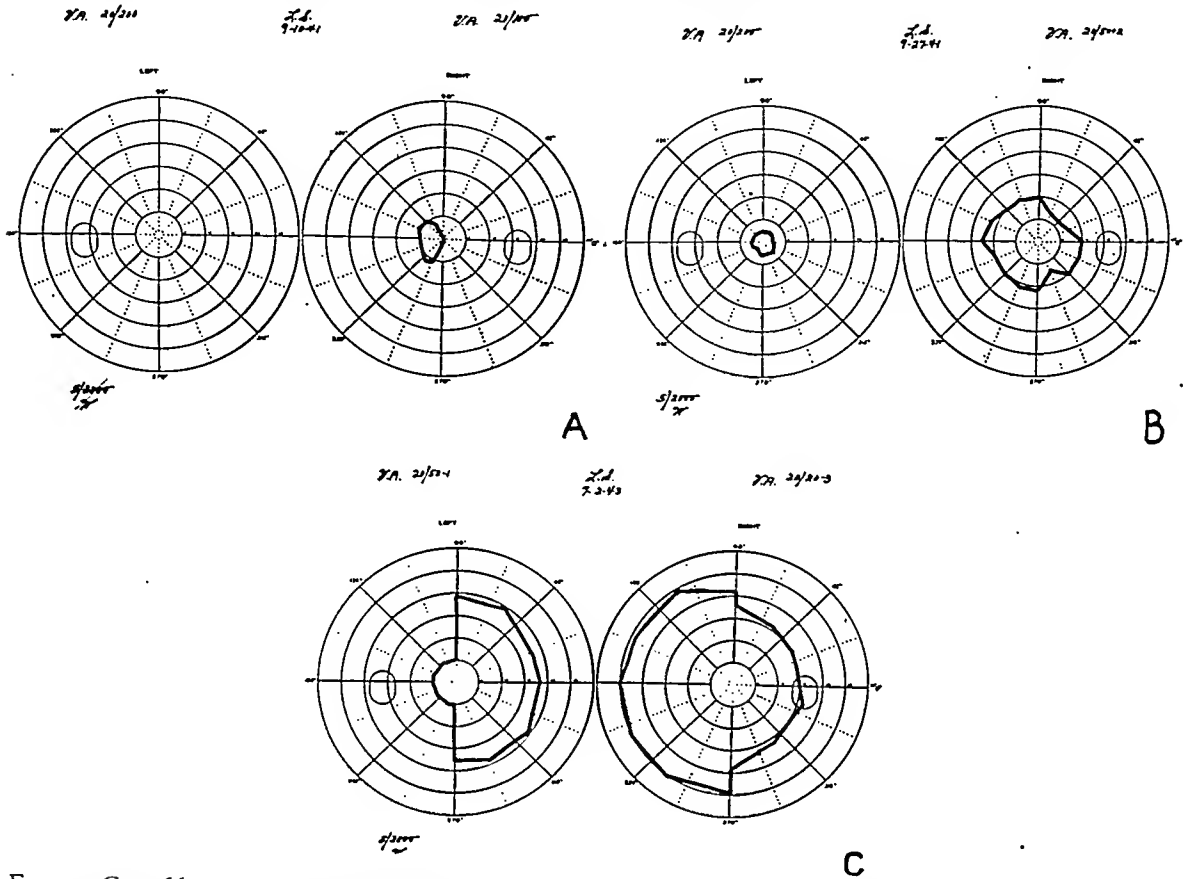
FIG. 13. Case 66. A, central fields prior to operation; B, central fields following operation; C, same, two years after operation and postoperative irradiation.

cisterna interpeduncularis. (Fig. 2.) In view of the fact that the sella was not enlarged, operation for purposes of verifying the nature of the lesion appeared indicated. On February 3, 1947, a right transfrontal craniotomy was performed, a chiasmal tumor encountered and a radical intracapsular removal accomplished. Examination of the tissue revealed it to be a chromophobe adenoma. Postoperative radiation therapy was given from February 26 to March 18, 1947, the amount being 1,000 r to each of three portals. Since then, visual acuity, central and peripheral fields have shown progressive improvement. Visual acuity at present is 15/13 on the right and 15/13 − 2 on the left. Central fields show slight remains of a defect in both upper temporal regions. (Fig. 12.) The patient is asymptomatic.

Examination revealed an obese woman with a smooth skin and a paucity of axillary and pubic hair. Visual acuity was 20/100 on the right and 20/200 on the left. A bitemporal hemianopsia was demonstrable grossly. Her basal metabolism was −27 per cent. Roentgenograms of the skull revealed enlargement of the sella turcica with destruction of the dorsum sellae and posterior clinoids. In view of the marked visual loss, operation for the purpose of decompressing the optic chiasm was deemed urgent. Accordingly on September 12, 1941, a right transfrontal craniotomy with intracapsular partial removal of a pituitary tumor was performed. The histologic diagnosis of the tumor was a chromophobe adenoma. At the time of discharge from the hospital on September 29, 1941, visual acuity and central fields

had improved in the right eye. In October, 1941, there was beginning improvement in the left eye also. Two courses of radiation therapy, each totalling 3,000 r, were administered postoperatively in October and December, 1941. In June, 1942, another course of irradiation was

left eye. Visual fields showed a suggestive upper temporal slant for color in the right eye. Funduscopic examination revealed no pathologic changes. Skull roentgenograms were reported to have shown enlargement of the sella turcica with thinning of the sellar floor and

Visual Acuity

Right Eye: Continuous line
Left Eye : Broken line
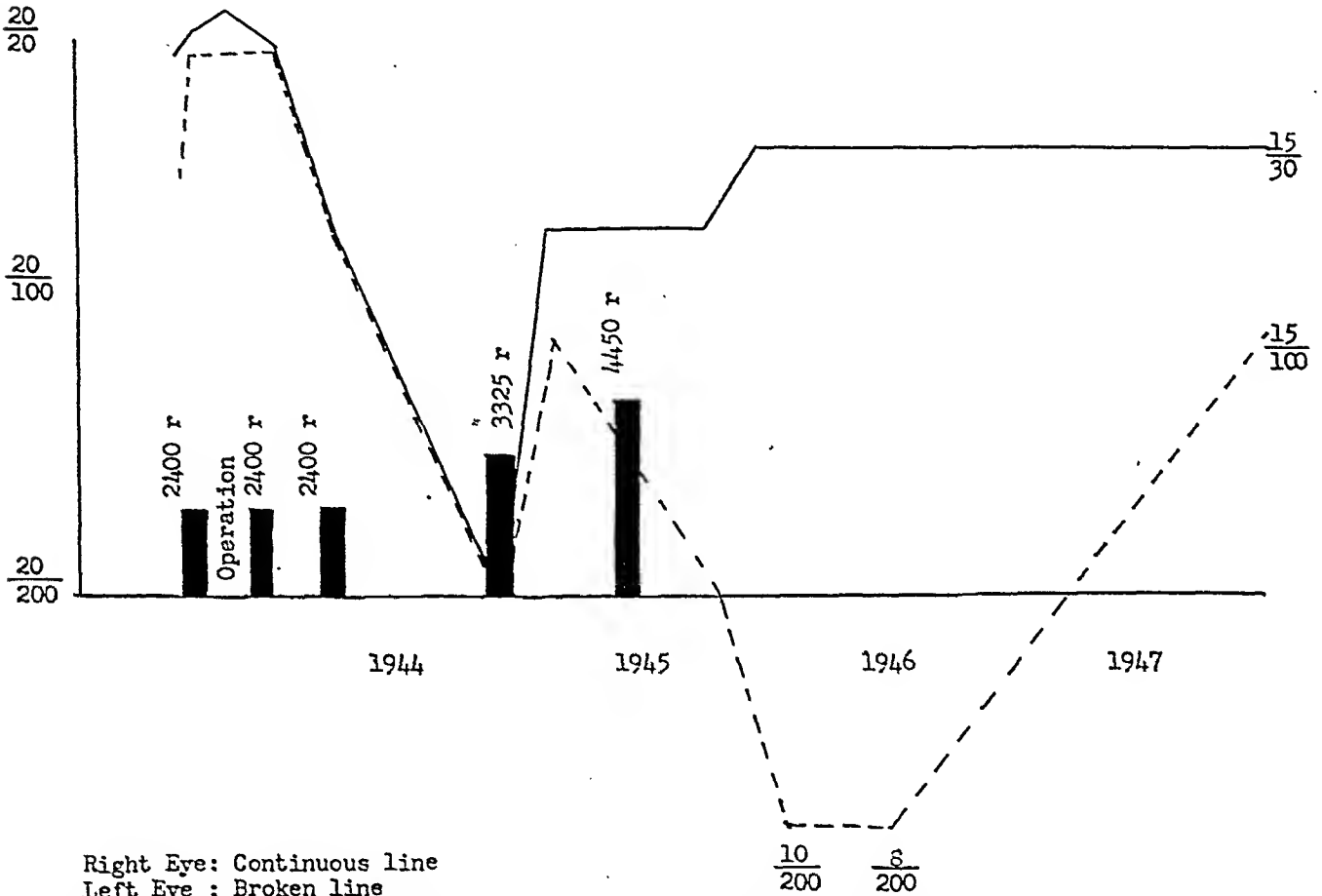
FIG. 14. Case 59. Graph showing the fluctuation of visual activity in each eye in relation to roentgenologic and surgical treatment.

given, the dose again being 3,000 r. Visual acuity in the right eye improved to 20/20 − 3 and in the left eye to 20/50 − 1. (Fig. 13.) The patient was last seen in July, 1947, at which time her vision was essentially unchanged, and except for an occasional headache, she was feeling well.

Radiation Therapy Pre- and Postoperatively with Varying Effects. I. S., (Case 59), Jewish Hospital, No. 264399. A fifty-two year old white male was admitted on May 2, 1943, because of diminishing vision of three years' duration. He had been examined in 1940 by an ophthalmologist who found a visual acuity of 20/20 in the right eye and 20/30 + 4 in the

erosion of the dorsum sellae and posterior clinoid processes. A pituitary neoplasm was suspected. In 1942, the patient began to experience severe headache, confined mainly to the right side. Visual acuity had become more severely affected since January, 1943.

Examination revealed a tall, thin man without any evidence of hypopituitarism. Bilateral optic atrophy of moderate degree was noted. Visual acuity on the right was 20/30 − 1 and on the left 20/50 − 1. Visual field examination revealed an unusual picture. There was an inferior altitudinal defect on the right and an inferior nasal slant on the left side. Roentgenograms confirmed the findings previously noted.

During May, 1943, a course of radiation therapy was given and 2,400 r applied. Visual acuity improved from 20/30 − 1 to 20/20 in the right eye, and from 20/50 − 1 to 20/30 + 2 in the left eye. The visual fields remained unaltered. It seemed most probable that the

was 20/70 bilaterally. Between May 31, 1944, and July 3, 1944, a fourth course of roentgen treatment was prescribed and 3,325 r administered. In June, 1944, visual acuity was 20/200 in both eyes. Very shortly thereafter, visual acuity began to improve and in September,
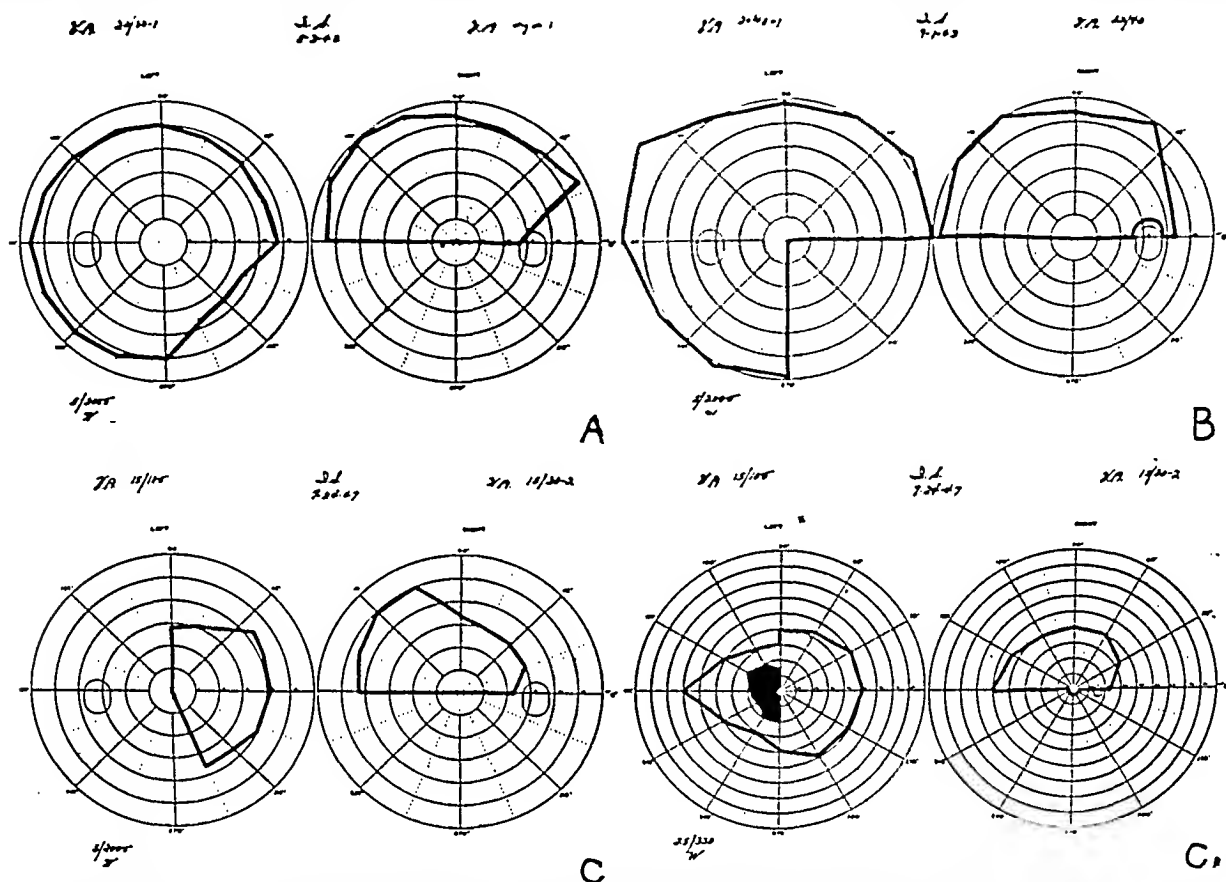
FIG. 15. Case 59. A, central fields prior to treatment; altitudinal hemianopsia; B, central fields following operation and postoperative irradiation; C-C1, most recent central and peripheral fields. Persistence of inferior hemianopsic defect.

underlying lesion was a pituitary adenoma, but in view of the incongruous visual fields operation was advised. On June 17, 1943, a right transfrontal craniotomy was performed and a pituitary adenoma disclosed. An extension of the tumor was observed to compress and displace the right optic nerve upward, thus, in all probability, accounting for the inferior visual field defect. Histologic examination of the tissue confirmed the diagnosis of a chromophobe adenoma.

Postoperative radiation therapy was instituted and a course of 2,400 r completed on July 15, 1943. Visual acuity, however, proceeded to decline. It was 20/40 in both eyes on September 1, 1943. In October, 1943, another course of radiation therapy was instituted and 2,400 r applied. On December 29, 1943, visual acuity

1944, measured 20/70 in the right eye and 15/100 in the left eye. Subsequently, however, regression of acuity of vision in the left eye occurred. Between November 27, 1944, and January 12, 1945, a fifth course of irradiation was given, and 4,450 r applied. Further improvement occurred in the right eye but the deterioration of vision in the left eye continued. It reached a low level of 8/200 in February, 1946, and thereafter began to improve.

When examined on July 24, 1947, visual acuity was 15/30 − 2 in the right eye and 15/100 in the left eye. (Figs. 14 and 15.) Hypopituitary stigmas were evident. The patient was pale, his hair very thin and libido was absent. He had been receiving 25 mg. of testosterone once a week for a period of two years. The only effect of this medication was to
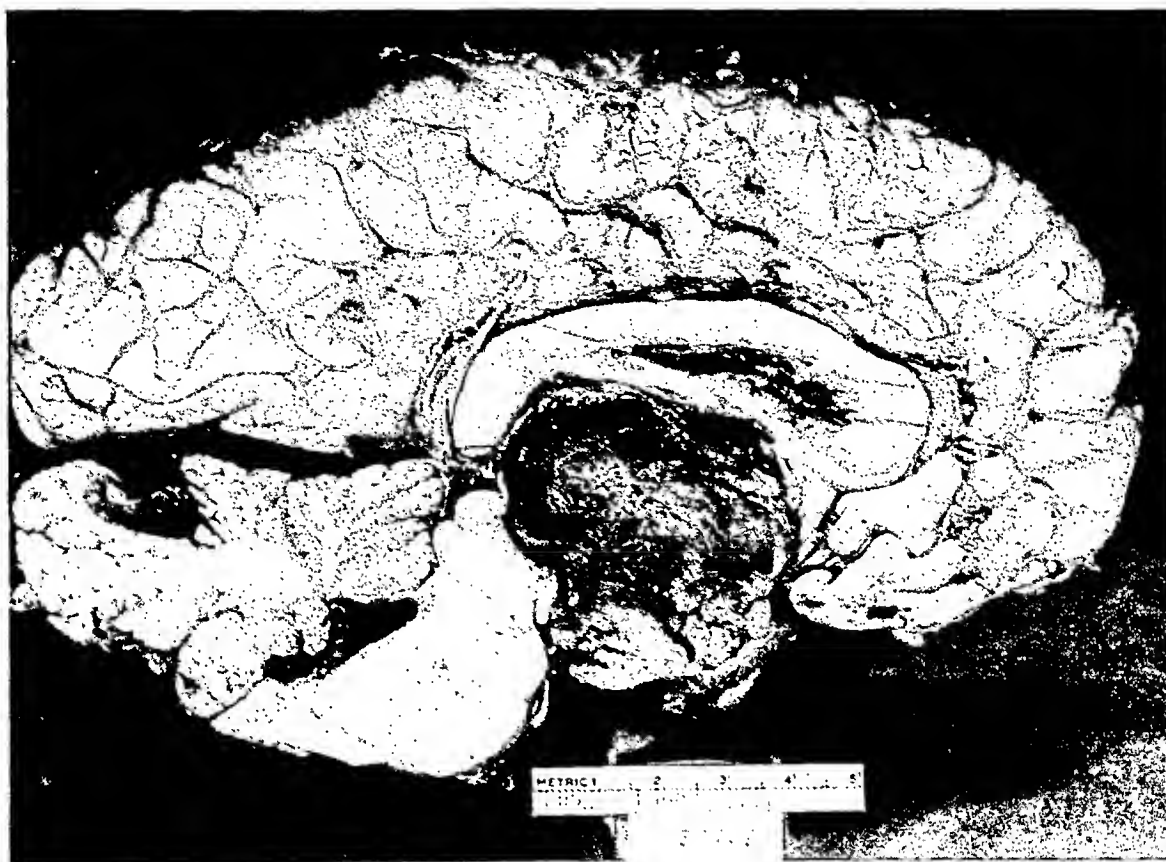
FIG. 16. Case 87. Sagittal section showing the tumor behind the optic chiasm projecting up into the third ventricle.

lessen his feeling of fatigue. He complained of no headache and was working at his occupation of a civil engineer.

Atypical Cases. R. C. (Case 87), Jewish Hospital, No. 210323. A fifty year old white male was admitted on March 21, 1938, because of impairment of memory and generalized weakness of one and a half year's duration. He had also begun to manifest emotional changes, consisting of profound depression and episodes of weeping.

Neurologic examination revealed a slight tremor of the extended fingers, some slowness of speech and possibly pallor of the temporal halves of the optic discs. Visual field studies were not altogether satisfactory but were suggestive of a bitemporal hemianopsic defect. Visual acuity measured 20/40 in the right eye and 20/30 in the left eye. Roentgenograms of the skull were negative; the sella turcica was not enlarged or atrophic.

The preponderance of mental symptomatology suggested a presenile type of degenerative disease. However, it was learned that the patient had been hospitalized elsewhere and a pneumencephalogram performed. The films showed a deformity of the cisterna interpeduncularis by a mass which also projected into the third ventricle. (Fig. 3.) A suprasellar tumor, possibly a meningioma, was considered.

On March 24, 1938, a right transfrontal craniotomy was performed and the region of the chiasm explored. Both optic nerves and the diaphragm of the sella turcica were well visualized but no tumor was seen.

The patient failed to recover postoperatively and expired the same day. Postmortem examination revealed a large tumor between the legs of the cerebral peduncles, extending upward and deforming the floor of the third ventricle. (Fig. 16.) The mass was behind the optic chiasm in a location which rendered it surgically unapproachable. Though entirely intracranial, the tumor was of pituitary origin since histologically it revealed the structure of a chromophobe adenoma.

J. S., (Case 86), Jewish Hospital, No. 255405. A fifty-seven year old white male was admitted on June 29, 1942, because of headache, impairment of gait, urinary incontinence and drowsiness. About five years previously, he had first experienced drowsiness but was nevertheless able to continue to work up until fifteen months before. At that time the drowsiness became more marked and, in addition, he became

unable to walk and developed headache. During the past three months, he was incontinent of urine.

Examination revealed a well nourished, drowsy, apathetic patient, responding only after much prompting. Orientation and mem-
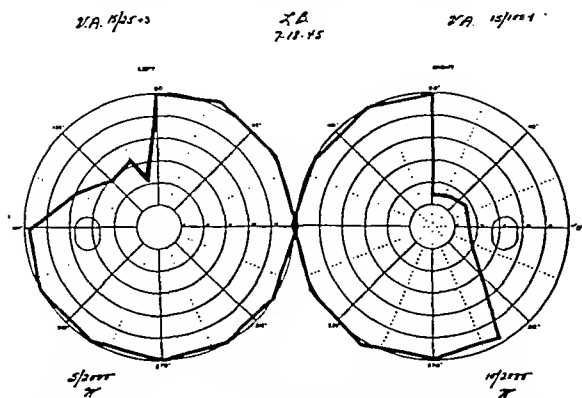
FIG. 17. Case 77. Central fields showing bitemporal hemianopsia, more pronounced on the right.

ory were defective. Objects were named correctly. The right pupil was a trifle larger than the left; both reacted sluggishly to light. No extraocular muscle weakness or conjugate gaze impairment was detected. There was a right central facial weakness with hyper-reflexia and a positive Babinski toe sign on the right. Sensory examination was unreliable. The fundi showed hazy disc margins. Visual field studies and determination of visual acuity were not feasible.

A ventriculogram was performed on June 30, 1942. This revealed considerable dilatation of the lateral ventricles and a deformity of the anterior portion of the third ventricle outlining a tumor mass. Concave filling defects in the floors of the frontal horns and an elevation of the cisterna interpeduncularis were also demonstrable. (Fig. 5.) The films showed, in addition, marked enlargement of the sella turcica with destruction of its bony boundaries. These findings were interpreted as being indicative of a tumor reaching into the third ventricle, possibly originating from the pituitary. A right transfrontal craniotomy extending across the midline was performed. Between the legs of the optic chiasm a mass was exposed and partially removed. It was evident that insufficient tumor could be removed through this approach. The mesial aspect of the hemisphere was, therefore, retracted, and after incising the corpus callosum, the lateral ventricle was entered. Tumor tissue was seen projecting through the foramen

of Monro. A considerable amount was removed, relieving the plug in the foramen and exposing the floor of the third ventricle. Histologic examination of the tissue revealed it to be a chromophobe adenoma.

The patient failed to recover from the operation and expired on the following day.

L. B., (Case 77), Jewish Hospital, No. 285402. A forty-three year old white female was admitted on July 16, 1945, because of headache, diplopia and impairment of vision of the right eye. Her illness began abruptly on May 18, 1945, with intense headache, drooping of the right lid and double vision. She was admitted to another hospital on May 21, 1945, where examination revealed ptosis of the right lid, paralysis of upward and inward movement and weakness of outward movement of the right eye. There was questionable diminution of pinprick appreciation over the ophthalmic division of the right trigeminal nerve. Mentally she was lethargic. Blood pressure was 165/85 mm. mercury. Red blood count was normal; the white blood count was 16,800, with a preponderance of polymorphonuclear leukocytes. A lumbar puncture done on May 21st revealed uniformly bloody cerebrospinal fluid containing over 4,000 red blood cells per cu. mm. The protein content of the cerebrospinal fluid was 90 mg./100 cc. The blood and cerebrospinal fluid Wassermann were both negative. Roentgenograms of the skull showed an enlargement of the sella turcica with thinning of the posterior clinoid processes. Visual fields revealed a constriction of the right temporal field. Electroencephalographic studies were essentially normal. While still in the hospital, the ocular palsies began to improve, but concomitantly progressive bilateral papilledema with hemorrhages appeared. The diminution of sensation over the first division of the right trigeminal nerve became more definite. Mentally, however, she was more alert. On June 22, 1945, the abnormal neurologic findings consisted of bilateral papilledema, a right partial ptosis, a smaller right pupil, weakness of elevation of the right eye, a right temporal field defect and diminished sensation over the right opthalmic distribution.

In view of the sudden onset of an ophthalmoplegia together with subarachnoid bleeding, the question of an aneurysm was given consideration. The combination of progressive papilledema together with disappearance of the

focal ocular palsies, however, was thought to be unusual for an aneurysm.

When admitted to this clinic on July 16, 1945, her neurologic status was unchanged from that previously noted except for the additional finding of weakness of abduction of the right eye. Visual field studies indicated a bitemporal hemianopsia, more marked on the right. (Fig. 17.) The visual acuity was 15/100 −1 in the right eye and 15/25 +3 in the left eye. The impression gained from the plain films of the skull was that the sella turcica was not enlarged but that there was marked atrophy of the dorsum sellae, sellar floor and posterior clinoid processes and that these changes were due to increased intracranial pressure. A ventriculographic study was carried out and this revealed slight dilatation of both lateral ventricles, without displacement, elevation of the anterior aspect of the third ventricle, and a tumor arising from the chiasmal region protruding into the cisterna interpeduncularis. On laminagraphic section, the tumor was seen to erode the anterior wall and floor of the sella turcica and to fill the sphenoidal sinus; enlargement of the sella turcica was clearly apparent. (Fig. 4.)

On July 19, 1945, a right transfrontal craniotomy was performed and a mass was seen presenting itself between the optic nerves. A subtotal removal was accomplished with adequate decompression of the optic chiasm. Histologic study of the tumor revealed it to be a chromophobe adenoma.

The patient improved considerably postoperatively and the papilledema slowly disappeared. In September, 1946, her neurologic status was negative. She complained of occasional headache and diplopia when very tired. Visual field studies on September 18, 1947, still showed bitemporal defects. The visual acuity measured 15/20 +2 in both eyes.

CHROMOPHILE ADENOMAS

In contrast to the number of chromophobe adenomas that came under observation, the cases with acromegaly were much fewer. There were thirty-five eosinophilic tumors so that the proportion of chromophobe to chromophile neoplasms in this series is about 3:1. The sex distribution was not especially unusual, twenty cases having occurred in females and fifteen in men. In general, the patients with acromegaly sought medical advice at an earlier age than did those with chromophobe tumors. Twenty-four of the thirty-five patients were in the fourth decade of life or younger when first seen. Of the remainder, nine were in the fifth decade. Although it was frequently difficult to establish with certainty the date of onset of either illness, the impression gained was that patients with eosinophilic adenomas manifested initial symptoms at an earlier age than did those with chromophobe tumors. Overgrowth of acral parts, which characterizes the disease, was present to varying degrees in all cases. A history was elicited of enlargement of the hands and feet made apparent by the necessity of procuring gloves and shoes of increasingly larger size, of growth and greater prominence of the lower jaw, together with separation of the teeth, of increase in size of the nose, lips, brows and tongue, resulting in the characteristic coarse facial features, and of thickening and swelling of the fingers.

In addition to symptoms of acral enlargement, a very common complaint was headache. It was present in twenty-two cases and often constituted the most distressing feature of the patient's illness. Amenorrhea or oligomenorrhea occurred in fifteen of the twenty female patients. In a few instances, normal menstrual periods were established following treatment. This took place in one patient following radiation therapy; in another, four years after operation and postoperative irradiation. The latter patient subsequently bore a child. In a third case, menses occurring regularly at intervals of two months were established following operation. Menstrual periods were resumed in still another patient after treatment with female sex hormone. Among the fifteen male patients, there were nine with diminution of libido, potency or both. One patient, whose libido was practically nil, reported striking improvement following the use of methyl testosterone. Potency was improved in another case following radiation therapy to

the pituitary. In comparison with the group of patients with chromophobe adenomas, the incidence of symptomatic visual impairment among the acromegalics was very low. It occurred in only six patients, one of whom was found to have papilledema and vascular retinal changes in association with hypertension. Two patients complained of transient diplopia but in neither did the examination reveal an extraocular muscle weakness. A bitemporal hemianoptic defect was demonstrable in one, and a very questionable similar defect in the other. Overgrowth of hair and excessive perspiration were mentioned in the protocols of about one-third of the cases although the actual frequency of their occurrence may have been greater. A variety of other symptoms was elicited. These included generalized weakness, easy fatiguability, joint pains, backache, gain or occasionally loss of weight, polydipsia and polyuria (in association with diabetes mellitus), somnolence, insatiable appetite, propensity for sweets, deepening of the voice and paresthesias.

The characteristic bodily changes, resulting from acral overgrowth and diagnostic of the disease, have already been mentioned. Studies of the visual fields indicated defects in seven cases. A bitemporal hemianopsia was demonstrated in six patients and a homonymous hemianopsia in one. Optic atrophy was observed in four of the cases. The patient with papilledema had ventriculography performed with negative findings, thus excluding an extrasellar tumor. The funduscopic changes were, therefore, interpreted as indications of a hypertensive neuroretinopathy.

Roentgen examination of the skull in these acromegalics revealed thickening of the calvarium, overdevelopment of the paranasal sinuses, prognathism, and usually enlargement of the sella turcica with thinning and depression of the floor, and erosion and posterior displacement of the posterior clinoid processes and dorsum sellae. In five cases, however, the size of the sella turcica was within normal limits.

When roentgenograms of the extremities were available, tufting of the terminal phalanges of the fingers was often noted. An increased basal metabolic rate averaging $+24$ per cent was found in half the cases in which such an examination had been performed. There were a few instances, on the other hand, in which it was abnormally low. There was evidence of disturbed carbohydrate metabolism, either an elevated fasting blood sugar level or an abnormally high glucose tolerance curve in six patients. Hypertension was a complicating factor in four of the thirty-five patients.

Treatment. The same plan of treatment was adopted for the eosinophilic adenomas as was employed in the chromophobe cases. Whenever therapy was indicated, irradiation was prescribed; and only if it failed to achieve its purpose, was operation undertaken. The results obtained in the individual cases are recorded in Table XII.

Twenty-nine patients were treated initially with irradiation and of these, improvement, measured in terms of symptomatic relief and cessation of acral growth, was achieved in eighteen. There was questionable progression of the acromegaly in three patients although one who had complained of headache was relieved of this symptom. Eight patients were unimproved and operation performed in six. Improvement following radiation therapy was obtained in a patient with recurrent symptoms who had been operated upon twice previously. Thus, of a total of thirty patients treated with irradiation, unequivocally favorable results were achieved in nineteen or 63 per cent. Operation following unsuccessful radiation therapy resulted in a cessation of visual failure in one case and in improvement in two other cases, without visual impairment, one of whom received postoperative roentgen treatment. Headache persisted in two patients after operation. In one the headache was probably of functional origin. It is too soon after treatment to evaluate the results in another patient.

TABLE XII

CHROMOPHILE ADENOMAS—TREATMENT AND RESULTS

Case	Therapy	Course
1. I. T. (non-hospitalized case)........	Radiation therapy	Normal vision; relief of headache; ? progression of acromegaly; 1½ yr. follow-up
2. R. B. (non-hospitalized case)........	Radiation therapy	Very slight progression of acromegaly over a 20 yr. period; normal vision
3. B. G. (non-hospitalized case)........	Radiation therapy	Relief of headache and drowsiness; vision normal; cessation of acral growth prior to therapy; 3 yr. follow-up
4. H. R. (J. H. #276441).............	No indication for therapy	Jacksonian seizures; negative pneumencephalogram
5. H. B. (non-hospitalized case)........	Radiation therapy	Relief of headache; no progression of acromegaly; vision preserved; 3 yr. follow-up
6. R. C. (non-hospitalized case)........	Radiation therapy	Relief of headache; cessation of growth prior to irradiation; vision normal; 10 yr. follow-up since irradiation
7. S. R. (non-hospitalized case)........	Radiation therapy	Relief of headache; subsequent recurrence of headache, probably of non-pituitary origin; severe hypertensive; cerebral vascular accident
8. I. S. (non-hospitalized case)........	Radiation therapy	Headache, somnolence improved; no further progression of acromegaly; 1½ yr. follow-up; vision normal
9. S. M. (non-hospitalized case)........	Radiation therapy	Unimproved symptomatically; vision normal; advancing acromegaly; 3 yr. follow-up
10. V. K. (non-hospitalized case)........	Radiation therapy	Cessation of growth; normal menses restored; vision normal; 4 yr. follow-up
11. M. A. (non-hospitalized case)........	Radiation therapy	Bitemporal hemianopsia; maintained same vision; no further progression of acromegaly; improved symptomatically; 2½ yr. follow-up
12. R. B. (N. I. #22185-H).............	Radiation therapy	Questionable slight bitemporal defect; relief of headache; no further progression of acromegaly; recent visual fields normal; 13 yr. follow-up
13. H. S. (non-hospitalized case)........	Radiation therapy	Relieved of paresthesias; no further acral enlargement; 3 yr. follow-up
14. H. W. (non-hospitalized case)........	Radiation therapy	Relieved of headache; potency better; no progression of acromegaly
15. S. D. (non-hospitalized case)........	Radiation therapy	Symptomatically improved; no further acral enlargement; 3 yr. follow-up
16. J. J. (J. H. #262844).............	Radiation therapy	Bitemporal hemianopsia; diplopia and weakness relieved; feels fine (letter); 3 yr. follow-up
17. I. F. (non-hospitalized case)........	Radiation therapy	Relieved of headache and joint pains; 5½ yr. follow-up
18. E. P. (non-hospitalized case)........	Radiation therapy	Headache relieved; hands softer and more pliable; 9 mo. follow-up
19. A. L. (N. I. #29442).............	Radiation therapy	Bitemporal hemianopsia; relieved of headache and weakness; vision unchanged; 1 yr. follow-up
20. R. C. (J. H. #249469).............	None	Homonymous hemianopsia; severe hypertension with hypertensive neuroretinopathy; death from cerebral hemorrhage
21. E. U. (non-hospitalized case)........	Radiation therapy	Relieved of joint pains; no progression of acromegaly; feeling well; 9 yr. follow-up
22. M. Y. (non-hospitalized case)........	Radiation therapy	Relatively asymptomatic for 12 yr.; recurrence of headache, drowsiness, further acral enlargement; vision unimpaired
23. P. W. (M. H. #27280-R)...........	Radiation therapy	Relieved of headache; no further progression of acromegaly; 11 yr. follow-up
24. C. Z. (non-hospitalized case)........	Transsphenoidal operation by Dr. Cushing in 1912	No headache since operation; no change in features for many years; normal vision; two children, aged 15 and 10; diabetic; 35 yr. follow-up

TABLE XII (Continued)

Case	Therapy	Course
25. M. A. (non-hospitalized case).......	Radiation therapy	Symptomatically improved; slight regression of acromegaly; 1 yr. follow-up
26. F. C. (non-hospitalized case).......	No indication for therapy	Asymptomatic; no recent progression of acromegaly; vision intact
27. B. S. (non-hospitalized case).......	Radiation therapy	Asymptomatic; questionable further growth of hands recently; further radiation therapy; 7 yr. follow-up
28. P. K. (J. H. #239283).............	Radiation therapy; operation	Persistence of headache despite irradiation; no visual disturbances; operation; postoperative recurrence of headache; further irradiation; relief of headache; improvement in features, return of menses; had a baby; 6 yr. follow-up
29. H. B. (M. H. #41602).............	Radiation therapy; operation	Headache and further acral growth despite irradiation; (6 courses over 9-year period); normal vision; operation; slight headache; 7 mo. postoperative
30. E. L. (M. H. #40150).............	Radiation therapy; operation	No relief of headache with irradiation; further acral enlargement; vision normal; operation; still having headache; 1 yr. postoperative; no increase in acromegalic features
31. M. G. (J. H. #241271)............	Radiation therapy; operation	No relief of headache with irradiation, nor following operation and postoperative radiotherapy; neurotic; no progress of acromegaly, nor visual impairment over 11 yr. follow-up period
32. P. W. (J. H. #266421).............	Radiation therapy; operation	Headache not relieved by irradiation; operation; improved; menses every 2 mo; vision normal.
33. J. R. (non-hospitalized case).......	Operation elsewhere via transphenoidal route; radiation therapy	Bitemporal hemianopsia; vision improved postoperatively; no evidence of further progress, 6 yr. after operation; headache due to associated hypertension
34. W. B. (non-hospitalized case).......	Radiation therapy; operation elsewhere	Bitemporal hemianopsia; visual impairment unrelieved by radiotherapy; operation; vision postoperatively about the same; feels fine 7 yr. postoperatively (letter)
35. M. M. (J. H. #272168).............	Two operations elsewhere; radiation therapy	Bitemporal hemianopsia; operation in 1933 because of visual impairment; recurrence of symptoms and reoperation in 1937; recurrence of visual symptoms and further acral growth in 1943; radiation therapy; no further progression of acromegaly; no further deterioration of vision; 3½ yr. follow-up; postoperative seizures

MALIGNANT ADENOMA—CASE REPORT

There was one instance of a malignant adenoma in this series.

G. O., Montefiore Hospital, No. 40337. An eighteen year old boy was first seen in this clinic on March 22, 1946, because of headache and diminished vision in the right eye. He had previously consulted a physician in 1944 at the age of sixteen because of failure to grow in the preceding four years. Physical examination at that time was essentially negative. He was 59 inches (147.5 cm.) in height. Basal metabolic rate was −11 per cent. Roentgen examination of the sella turcica was reputedly negative. Thyroid medication was prescribed. In January, 1945, he began to complain of headache relieved by vomiting. This headache continued intermittently and in January, 1946, an ophthalmologist was consulted who found evidence of optic atrophy. In the period of a year, the patient had gained 22 pounds and grown 1 inch. Roentgen study of the skull at this time revealed marked enlargement of the sella turcica with thinning of the posterior clinoids. There was no suprasellar calcification. Basal metabolic rate was −21 per cent. A glucose tolerance

test revealed a normal curve. The fasting blood sugar was 139 mg. per cent.

Examination in this clinic on March 22, 1946, revealed a short, pale, obese boy with a Froehlich type of body build. The genitalia were small, pubic and axillary hair scanty, and the hair on his head of a silk-like quality. Both optic discs showed considerable atrophy. Visual acuity was 15/30 in the right eye and 15/70 in the left eye, not improved by correction. Perimetric examination revealed constriction of the left visual field, with a depression in the upper temporal field, and a right temporal hemianopsia together with a defect in the right inferior nasal field. The clinical picture suggested a craniopharyngioma while the x-rays favored a pituitary adenoma or possibly an intrasellar craniopharyngioma.

On April 24, 1946, a right transfrontal craniotomy was performed. A cystic tumor medial to the right optic nerve was encountered and an incomplete removal accomplished. Examination of the tissue by Dr. H. M. Zimmerman revealed tumor cells of pituitary parenchymal origin showing pleomorphism and mitotic division. These cells invaded a thickened dural capsule. They appeared to be neither chromophilic nor ordinary chromophobe cells. The pathologic diagnosis was a pituitary carcinoma.

Unfortunately institution of radiation therapy was delayed. From November 29, 1946, to January 27, 1947, the patient received 1,000 r each to a right and left lateral portal and 825 r to a frontal portal. Visual loss has been progressive so that now vision in the right eye is limited to light perception. Visual acuity in the left eye is 15/20 but the field of vision has diminished considerably.

PITUITARY BASOPHILISM

Five patients were observed who appeared to fulfil the criteria for a diagnosis of Cushing's syndrome. Their ages were twenty-three, thirty, thirty-two, thirty-four and fifty-five years, respectively. Four of the five patients were females. All presented manifestations of the disease to varying degrees. The findings included adiposity of the face, neck and trunk (buffalo distribution), hirsutism, osteoporosis, sexual dystrophy, hypertension, purplish striae, polycythemia, kyphosis, a tendency to bruise easily, purpura, diminished resistance to cutaneous infection, acrocyanosis, edema of the lower extremities, slight exophthalmos and weakness.

A variety of therapeutic procedures were utilized. In the one case in which an adrenal tumor was found, this was removed. Irradiation of the pituitary was given a trial in three cases. In none did it appear to yield beneficial results. Radon seeds were implanted into the pituitary fossa of one patient. Although insufficient time has elapsed to evaluate fully the effects of this procedure, some improvement seems to have been achieved clinically. Two patients were also under the care of Dr. Fuller Albright who treated them with testosterone and potassium chloride. The testosterone was subsequently discontinued, and on the potassium chloride medication alone favorable results appear to have been obtained in one of these patients. Summaries of the five cases follow:

L. S., a thirty year old white male, a dentist by occupation, first came under observation on September 12, 1945, because of a multiplicity of symptoms. He was a known hypertensive since August, 1943. About that time, too, he had begun to develop a fulness in the region of his jowls and redness of the face. During the summer of 1945, he became aware of decreased libido though potency was retained. More recently he noticed some swelling of the lower extremities and a tendency to bruise easily. He gained 6 pounds and whereas his extremities remained unchanged, his face and abdomen had become more obese. In August, 1945, he developed large purple striae over the abdomen. There was also some diminution in the growth of his beard.

The significant findings on examination included a roundness of the face which was unusually florid, slight exophthalmos of the left eye, an acneiform eruption of the upper trunk, a slight buffalo hump, some pitting edema of both feet, purple striae over the lower abdomen and a blood pressure of 160/120 mm. mercury. The diagnosis of a Cushing syndrome was made and in December, 1945, he was referred to Dr. Fuller Albright at the Massachusetts General Hospital for further study.

There, detailed laboratory studies were performed, the results of which are listed below:

Hemoglobin........... 16.9 Gm./100 cc.
Red count............ 5.8 million
White count.......... 14,400 (86% polymorphonuclear leukocytes, 10% small lymphocytes, 3% monocytes)
Serum calcium........ 10.6 mg./100 cc.
Serum phosphorus..... 3.2 mg./100 cc.
Serum phosphatase..... 3.2 Bodansky units
Vitamin A............ 1.2 units/cc.
Carotinoids.......... 2.2 units/cc.
Cephalin flocculation... 24 hours—negative; 48 hours —negative.
Serum cholesterol...... 212.0 mg./100 cc.
Van den Bergh........ normal
Serum protein........ 6.96 Gm./100 cc.
Serum albumin........ 5.24 Gm./100 cc.
Serum globulin........ 1.72 Gm./100 cc.
A/G ratio............ 3.10
Serum N. P. N........ 27.0 mg./100 cc.
Serum sodium......... 145.9 mEq./L.
Serum potassium....... 3.9 mEq./L.
Serum chloride........ 105.0 mEq./L.
Serum CO_2 content.... 30.9 mEq./L.
Glucose tolerance test:
  Fasting............. 79 mg./100 cc.
  ½ hour............. 140 mg./100 cc.
  1 hour............. 140 mg./100 cc.
  2 hours............ 86 mg./100 cc.
  3 hours............ 94 mg./100 cc.
  4 hours............ 92 mg./100 cc.
Insulin tolerance test: (7.8 units insulin intravenously)
  0.................. 103.0 mg./100 cc.
  20 minutes......... 78.0 mg./100 cc.
  30 minutes......... 57.0 mg./100 cc.
  52 minutes......... 78.0 mg./100 cc.
  60 minutes......... 75.0 mg./100 cc.
  90 minutes......... 100.0 mg./100 c.
  120 minutes........ 99.0 mg./100 c:.
  45 minutes after 0.55 cc. of epinephrine 1/1000
                                    132 mg.
  60 minutes after epinephrine   144 mg.
Prothrombin time normal.
Urine culture: alpha hemolytic streptococci and Staphylococcus albus
Electrocardiogram normal
17-ketosteroid excretion: 21.0 mg./24 hours
Beta ketosteroid....... 1.1 mg./24 hours
11-oxysteroid excretion. 3.7 mg./24 hours
Intravenous pyelography revealed normal findings except that the left kidney was lower than the right.

X-rays of the skull and spine were normal. The findings of an elevated 11-oxysteroid excretion, high serum sodium level, low potassium content, high white blood count and low percentage of lymphocytes were in keeping with the diagnosis of Cushing's syndrome. There was marked variation in the degree of abnormality of these findings. His 11- oxysteroid excretion varied from 3.7 to 0.27 mg./24 hours;

17- ketosteroid excretion varied from 29 mg. to 9.2 mg./24 hours; the percentage of lymphocytes varied from 22 to 9 per cent.

The urinary infection responded to 5 Gm. of sulfathiazole daily. Symptomatic improvement was obtained with potassium chloride in 5 Gm. daily doses.

Perirenal air insufflation was non-contributory. On December 22, 1945, Dr. Oliver Cope explored the left adrenal gland. No tumor was found and biopsy showed a normal or hyperplastic gland.

The laboratory indices reached their most abnormal values on the second postoperative day. Thereafter steady improvement occurred. Thus 11- oxysteroid excretion was 5.0 on December 23rd, 2.6 on December 24th, 3.0 on December 25th, 1.6 on December 26th, 0.4 on December 27th, 0.46 on January 11th, 0.43 on January 17th, 0.48 on January 29th, 0.42 on February 4th, and 0.34 on February 10th. The percentage of lymphocytes rose from 9 on December 24th to 35 on February 9th.

Irradiation of the pituitary was begun on December 28, 1945, and completed on January 11, 1946. He received 900 r to each of four portals.

Metabolic studies revealed a positive nitrogen balance but a negative calcium balance. The addition of potassium chloride further increased the positive nitrogen balance and decreased to one-third the negative calcium balance.

He was discharged from the hospital on February 10, 1946, on a regimen of five 0.3 Gm. capsules of potassium chloride. When reexamined in March, 1946, he showed considerable improvement. All his signs appeared to be regressing. Cyanosis was much less pronounced. The jowls and heavy fat deposits in the clavicular and breast regions were appreciably reduced. Striae were still present, though much paler. He felt stronger and libido was definitely improved. In July, 1946, his blood pressure was 170/90 mm. mercury. Further laboratory studies by Dr. Albright revealed the following data:

White blood count...... 9,900
Differential count....... 73% polymorphonuclear leukocytes, 20% small lymphocytes, 5% monocytes, 2% eosinophiles
Urine culture.......... Occasional alpha hemolytic streptococci and Staphylococcus albus

17—ketosteroid excretion   17.2 mg./24 hours
11—oxysteroid excretion.   1.1 mg./24 hours
Serum sodium..........   149.1 mEq./L.
Serum potassium.......   5.3 mEq./L.
Serum chloride.........   106.0 mEq./L.
Serum CO₂.............   32.3 mEq. L.
Serum sugar...........   91.0 mg./100 cc.
Serum cholesterol......   287.0 mg./100 cc.
Total protein..........   7.06 Gm./100 cc.
Serum albumin........   4.84 Gm./100 cc.
A/G ratio.............   2.2
Serum N. P. N.........   31.0 mg./100 c.

The normal white blood count and absence of a marked lymphocytopenia indicated improvement. The blood pressure, sodium level and 11- oxysteroid excretion were still abnormal. Another course of sulfathiazole was prescribed, as well as continuation of the potassium chloride.

In October, 1946, the patient reported further improvement. He had been playing tennis and doing a full day's work in the office. His blood pressure was 170/100 mm. mercury. He weighed 192½ pounds (197½ pounds in June, 1946). Urine culture still showed a few staphylococcus albus and non-hemolytic streptococci colonies. Laboratory studies revealed a diminution in the sodium and cholesterol content of the serum and a normal 11- oxysteroid excretion.

White blood count......   13,000
Differential count.......   80% polymorphonuclear leukocytes, 16% small lymphocytes, 4% monocytes
Serum sodium..........   143.0 mEq./L.
Serum potassium........   4.6 mEq./L.
Serum chloride.........   104.0 mEq./L.
Serum CO₂.............   31.0 mEq./L.
Serum cholesterol.......   231.0 mg./100 cc.
17—ketosteroid excretion   21.2 mg./24 hours
11—oxysteroid excretion.   0.4 mg./24 hours

In January, 1947, another course of sulfathiazole therapy was administered because of persistence of a positive urine culture. At that time serum sodium, potassium, chloride and CO₂ were normal. The 17- ketosteroid excretions were 18.8 and 26.6 mg./24 hours; the 11- oxysteroid excretions 0.48 and 0.56 mg./24 hours. Daily injections of 25 mg. of testosterone were administered between May 17th and May 30, 1947. No improvement resulted from this treatment.

At present the patient feels quite well. His weakness has not reappeared; libido and potency are normal; blood pressure is 150/90 mm. mercury. His face is less round and florid. Purplish striae are still evident. Visual fields are full and visual acuity normal bilaterally. He continues to take 4½ Gm. of potassium chloride daily.

The most recent laboratory studies performed on May 31, 1947 revealed the following:

Serum sodium..........   140.0 mEq. L.
Serum potassium.......   4.5 mEq. L.
Serum chloride........   103.0 mEq. L.
Serum CO₂.............   28.4 mEq. L.
Urine culture..........   Moderate growth of staphylococcus albus; diphtheroid and B. coli
17—ketosteroid excretion   11.6 mg. 24 hours
11—oxysteroid excretion   0.65 mg./24 hours

B. C., Montefiore Hospital, No. 42543. A thirty-two year old white, Jewish female was referred to this clinic in March, 1947. She had been well till after the delivery of her second child in October, 1942, when her menses failed to return. Menstrual periods were resumed following estrogen therapy but ceased when this medication was discontinued. Some time in 1945 a redistribution of adipose tissue occurred so that her face, chest, abdomen and hips increased in size, whereas her arms and legs seemed thin in proportion. An increasing roundness and duskiness of her face was noted. There was no gain in weight, however. About this time, too, an elevated blood pressure was discovered. An increased growth of hair appeared over the upper lip and extremities. Her skin began to bruise easily and a marked pyodermia also developed. Weakness and fatiguability had become progressively more marked for a year and a half.

She was studied at another hospital where the diagnosis of Cushing's syndrome was made. There was no evidence of an ovarian or thymic lesion. Perirenal air insufflation was carried out, enlargement of the right adrenal gland suspected, and on July 31, 1946, an exploratory operation performed. No tumor was found but only what was thought to be a hyperplastic adrenal gland. Radiation therapy was instituted in November and December, 1946, and a total of 2,000 r administered to the pituitary region. No improvement resulted.

Examination in this clinic revealed a short, obese female with a rounded face and thin limbs. Her face and extremities were purplish-red in color and her chest covered with a maculopapular rash. There were ecchymoses and healed abrasions over both legs. A slight excess of hair involving especially the mustache area and the region about the nipples was

FIG. 18. Radon seeds are visible within the sella turcica; the latter is normal in size. There is a demineralization of the entire skull.

noted. No striae were seen. Blood pressure was 190/115 mm. mercury. Laboratory studies revealed the following:

Red blood count.......... 5.0 million
White blood count........ 11,650 (normal differential count)
Platelet count............ 232,000
Bleeding time............ 2½ minutes
Clotting time............ 3 minutes, 5 seconds
Prothrombin time
    Undiluted plasma....... 22.9 seconds
    Diluted plasma......... 49.6 seconds
Blood urea nitrogen....... 12.5 mg./100 cc.
Serum cholesterol......... 145 mg./100 cc.
Blood sugar............. 85 mg./100 cc.
Serum calcium........... 10.1 mg./100 cc.
Total protein............ 7.2 Gm./100 cc.
Albumin content......... 5.6 Gm./100 cc.
Globulin content......... 1.6 Gm./100 cc.
A/G ratio.............. 3.8
Glucose tolerance test
    Fasting................ 85 mg./100 cc.
    1 hour................ 296 mg./100 cc.
    2 hours............... 206 mg./100 cc.
    3 hours............... 49 mg./100 cc.
    4 hours............... 66 mg./100 cc.
Basal metabolism........ −16 per cent

Perimetric examination indicated no defects. Electroencephalography revealed an essentially normal pattern. The electrocardiogram showed a left axis deviation. Roentgen examination of the skull, spine and extremities indicated a moderate degree of demineralization; the sella turcica was not enlarged and there was no erosion of the clinoid processes.

Neoplastic disease of the adrenals having been excluded, a direct surgical attack on the pituitary gland was planned and on May 5, 1947, a right transfrontal craniotomy was performed. An anteriorly placed optic chiasm was visualized and it was immediately apparent that an attempt to incise the diaphragma sellae and remove tissue with a pituitary spoon would jeopardize the integrity of the chiasm. An opening was, therefore, made in the diaphragm with a lumbar puncture needle and five radon seeds, each containing 1.04 millicuries, implanted. (Fig. 18.) The patient withstood the operative procedure very well and her postoperative course was equally uneventful.

Since discharge from the hospital on June 2, 1947, the patient has been seen at frequent intervals. Subjectively she feels much improved. Objectively a few slight changes have occurred. Her dusky color is a trifle less marked and the contour of her face has tended to assume more normal proportions. Menses have not occurred. Her weight is unchanged over the preoperative figure (132 pounds). Blood pressure has varied between 135/85 and 170/110 mm. mercury.

Further observation will be necessary to evaluate the results of treatment in this case.

M. H., Montefiore Hospital, No. 41263. A fifty-five year old Jewish housewife was admitted on October 4, 1946, because of backache of several years' duration. Eight months ago a sensation of something snapping in her low back was experienced after bending, followed by a persistent, severe non-radiating pain. About two years previously, a change in her appearance had become apparent. She developed a puffiness of the face and neck and a prominence of the eyes. Her basal metabolism was found to be low and thyroid medication was administered. During the past year, she had been physically weaker. A splenectomy was performed twenty years ago for purpura hemorrhagica; purpuric spots were still evident occasionally.

She presented a striking appearance on physical examination. Her face was plethoric with heavy jowls, some telangiectases and a moderate degree of hirsutism on the sides of the cheeks and on the upper lip. Her extremities relative to her torso were thin. There were no striae present. The lumbosacral spine was tender and the Lasegue maneuver positive bilaterally. No neurologic abnormalities were demonstrable. Blood pressure was 115/85 mm. mercury. Laboratory data were as follows:

Red blood count	5.07 million
Hemoglobin	14.8 Gm./100 cc.
White blood count	9850 (75% polymorphonuclear leukocytes, 21% lymphocytes, 1% eosinophiles, 3% monocytes
Platelet count	268,000
Bleeding time	1½ minutes
Clotting time	2 minutes
Plasma prothrombin time	
Undiluted plasma	18 seconds
Diluted plasma	38 seconds
Blood sugar	112 mg./100 cc.
Blood urea nitrogen	15.8 mg./100 cc.
Serum cholesterol	279 mg./100 cc.
Serum chloride	343 mg./100 cc.
Serum calcium	10.6 mg./100 cc.
Serum sodium	331 mg./100 cc.
Serum phosphorus	3.4 mg./100 cc.
Alkaline phosphatase	3.9 units
Serum total protein	6.7 Gm./100 cc.
Albumin fraction	4.8 Gm./100 cc.
Globulin fraction	1.9 Gm./100 cc.
Glucose tolerance test	
Fasting	74 mg./100 cc.
½ hour	150 mg./100 cc.
1 hour	110 mg./100 cc.
2 hours	66 mg./110 cc.

Insulin tolerance curve	
Fasting	70 mg./100 cc.
20 minutes	41 mg./110 cc.
40 minutes	47 mg./100 cc.
60 minutes	161 mg./100 cc.
90 minutes	108 mg./100 cc.
45 minutes after adrenalin	155 mg./100 cc.
Urine examination	negative
Basal metabolism	−12 per cent
Sulkowitch test	positive
11—oxysteroid excretion	normal

Visual fields showed no defects. The electroencephalogram revealed an essentially normal pattern. Electrocardiography indicated a left axis deviation. Roentgenogram of the skull, thoracic cage and spine revealed a generalized osteoporosis; the sella turcica was otherwise normal.

Perirenal air injection revealed a distinctly enlarged adrenal gland on the right. The left adrenal gland could not be visualized on several occasions. It was, therefore, planned to explore the left side first and, if a normal adrenal gland was found, to remove the enlarged right one at a subsequent session. On January 29, 1947, the left suprarenal region was explored and an adrenal adenoma found and removed.

She continued to experience back pain postoperatively which was relieved by estrogen therapy. A thrombophlebitis of the left lower extremity followed by a pulmonary embolism complicated her subsequent course. After recovery from this complication, there was marked subjective improvement and she was able to get about without much pain. Prior to discharge from the hospital in August, 1947, her blood pressure ranged around 135/90 mm. mercury. Roentgenogram of her spine showed a collapse of the dorsal vertebrae 5, 6, 7 and 9. It was planned to fit her with a brace in the orthopedic clinic.

L. B., a thirty-four year old white female was seen on May 25, 1943, because of amenorrhea of about two years' duration and swelling of the face. An elevation in blood pressure had recently been discovered. She had always been inclined to have an excess of hair, perhaps more so lately. A tendency to bruise readily was also observed. She had had roentgenograms taken of her skull which were reported to be negative. Her basal metabolism was +2 per cent. She had been treated with thyroid extract, stilbesterol and aminophylline. The patient was married and had two children, one ten years old, the other six and one-half years of age.

Examination revealed a well developed female with a bloated face and jowls, facial hirsutism, prominence of the eyes, mottling of the skin, areas of ecchymoses, protuberance of the abdomen, slight edema of the feet and a buffalo hump. Blood pressure measured 190/130 mm. mercury. She weighed 110 pounds although she stated her usual weight fluctuated around 103 pounds.

Cushing's syndrome was diagnosed and radiation therapy to the pituitary instituted. Between June and October, 1943, three courses of irradiation, each totalling 2,400 r, were administered. The patient reported no change in her condition following this treatment.

She was subsequently under the care of Dr. Fuller Albright at the Massachusetts General Hospital during the spring and summer of 1946. Extensive studies were carried out, the results of which afforded confirmatory evidence of the diagnosis of Cushing's syndrome. Thus, low serum phosphorus and potassium levels, a high serum carbon dioxide content and an elevated 11-oxysteroid excretion were found. The insulin tolerance test was, however, normal. There was generalized osseous decalcification. The electrocardiogram indicated a left axis deviation. Treatment consisted of 25 mg. of testosterone proprionate every other day and 8 Gm. of potassium chloride daily. A Bacillus coli urinary infection responded to streptomycin. Perirenal air studies revealed no definite abnormalities. Exploration of the left adrenal gland revealed no tumor. Biopsy showed a normal or hyperplastic gland. It was deemed advisable to explore the right adrenal gland at a subsequent session but permission to do so was not granted. Despite the large intake of potassium, her serum potassium remained low.

The patient was seen again on September 25, 1947, at which time she was feeling quite well. Her weight was 109½ pounds. She still bruised easily and her physical findings remained unchanged. Blood pressure was 190/140 mm. mercury. Menses were reestablished in September, 1946, and had been regular in occurrence since then. She was still taking 6 Gm. of potassium chloride daily.

M. G., Jewish Hospital, No. 218322. The original example of the syndrome of pituitary basophilism described by Harvey Cushing in 1912 as a "polyglandular syndrome of painful obesity, hypertrichosis and amenorrhea with overdevelopment of secondary sex characteristics" and subsequently reclassified as a basophilic adenoma, came under the senior author's observation in 1939. Being of unusual interest from the historical as well as from a clinical standpoint, her history will be briefly reviewed and the observations recorded in 1939 added.

A twenty-three year old unmarried woman of Jewish extraction was admitted to the Johns Hopkins Hospital on December 29, 1910, because of amenorrhea since the age of sixteen, increasing obesity, headaches, nausea and vomiting. Her weight had increased from 112 to 137 pounds in the two years prior to admission. Other symptoms included pain in the eyes, insomnia, tinnitus, extreme dryness of the skin, frequent sore throats, dyspnea, palpitation, purpuric outbreaks, recurring nose bleeds and marked constipation with bleeding hemorrhoids. Growth of hair had appeared on the face. She had become increasingly round-shouldered. There was marked weakness and constant backache and epigastric pain. Examination revealed a short, kyphotic woman, with a round, dusky face which had an excess of hair. Adiposity affected the face, neck and trunk and relatively spared the extremities. The body and lower extremities were especially cyanotic and the skin bruised easily. There was considerable pigmentation. Skull films revealed a normal sella turcica. The red blood count was 5.3 million, the white count 12,000, and the hemoglobin 85 per cent. Systolic blood pressure averaged 185 mm. mercury. In September, 1911, a subtemporal decompression was performed because of continued headache.

She was again studied at the Peter Bent Brigham Hospital in 1913 because of further increase in weight (151 pounds). Her symptoms and general condition were essentially unchanged. Blood pressure averaged 180/110 mm. mercury. There was still a slight polycythemia, the red blood count being very slightly in excess of 5 million.

Late in 1913 menses were resumed, though irregularly. They continued until about the age of forty-six. In 1917, an operation for a renal calculus was performed at another hospital.

In November, 1922, she was readmitted to the Peter Bent Brigham Hospital. Her blood pressure ranged around 160/95 mm. mercury, the red blood count was 5.24 million, the basal metabolic rate was −9 per cent. Her general appearance remained unaltered, although there

had been some loss of weight. Skull films showed a generalized demineralization.

She came under Dr. Cushing's observation again in February, 1932, at which time many of her old stigmas were no longer evident. Her weight was 105 pounds and her blood pressure normal. She no longer bruised easily, her plethora was gone and her striae pale instead of purplish. The red blood count was 5.19 million and hemoglobin 111 per cent (Sahli). Her kyphosis was more pronounced.

In January, 1939, she was admitted to the Jewish Hospital of Brooklyn because of pain in the left flank radiating to the left lower quadrant. She had passed six or seven calculi in her urine during the previous ten years. Retrograde pyelography revealed no evidence of a stone, nor of other renal abnormalities. Physical examination showed slight facial hirsutism, a relaxed, pendulous abdomen, remains of the old striae, thin but not disproportionate extremities. She weighed 117 pounds; her blood pressure was 130/80 mm. mercury. Laboratory studies revealed the following:

Hemoglobin	88 per cent
Red count	5.0 million
White count	5500 (52% polymorphonuclear leukocytes, 43% lym phocytes, 2% monocytes, 3% eosinophiles)
Serum calcium	12 mg./100 cc.
Serum phosphorus	2.9, 3.2 mg./100 cc.
Serum phosphatase	4.8 units
Blood sugar	87 mg./100 cc.
Blood urea nitrogen	16.8 mg./100 cc.
Blood uric acid	5.6 mg./100 cc.
Phenol sulphonphthalien excretion	57.8% (2 hours)
Urine	Trace of albumin

COMMENTS

Diagnosis. The diagnosis of a pituitary tumor, either of the chromophobe or chromophile type, ordinarily offers little difficulty. As a rule, both varieties are diseases of adult life. Patients suffering from chromophobe adenomas are much more frequently encountered; and usually when they reach the neurosurgeon, the outstanding symptom is impairment of vision. Acromegalics, on the other hand, seek advice primarily because of headache and also because of the striking alterations of their features. Manifestations of hypopituitarism are usually evident in the chromophobe group and include such signs and symptoms as pallor and atrophy of the skin, loss of hair, depression of sexual function, gain in weight, a lowered basal metabolic rate and a tendency toward somnolence. The characteristic bodily changes produced by an eosinophilic adenoma render the diagnosis obvious at a glance in a well marked case. The head is large, the jaw prognathic, the nose prominent, the tongue thickened and the teeth widely separated. The extremities are large, the shoulders rounded and the hands spade-like and puffy. Among the numerous complaints are headache, amenorrhea, diminished libido and potency, paresthesias, excessive perspiration and joint pains. Diabetes mellitus is not an infrequent occurrence in this disease. Many other signs and symptoms may be present, as indicated by Davidoff[6] in a review of 100 cases of Dr. Cushing's series. Occasionally, signs of hypopituitarism coexist with evidence of preexisting hyperpituitarism. The term "fugitive acromegaly" has been applied by Bailey and Cushing[3] to such mixed cases.

Visual disturbances resulting from pressure on the optic chiasm and nerves are exceedingly common, especially in the group of chromophobe adenomas, and are of the utmost diagnostic importance. The demonstration of a bitemporal hemianopsia clearly indicates a lesion involving the optic chiasm. This is the most constant visual field disturbance produced by a pituitary tumor. For the detection of early visual field changes, the use of the tangent screen at a distance of two meters, together with small test objects, is far superior to ordinary perimetry. Usually the earliest indications of chiasmal compression are defects in the superior temporal quadrants. With further growth of the tumor, a complete bitemporal hemianopsia results; and if the process continues to remain unchecked, the inferior nasal fields are affected and finally vision fails altogether. Primary optic atrophy accompanies these changes. This is the usual sequence of events. Occasionally, however, a visual defect other

than a bitemporal hemianopsia is observed. Thus, these tumors may implicate the optic tract on one side, in which case a homonymous hemianopsia is produced. Should the optic chiasm happen to be located further posteriorly than usual, only one optic nerve may be involved and the symptoms be referable to but one eye.

Confirmatory roentgenographic evidence of an intrasellar tumor, consisting of enlargement and atrophy of the sella turcica, is present in the vast majority of cases of chromophobe adenoma. To a somewhat lesser extent this is also true of the eosinophilic tumors. The first structures to be involved are the dorsum sellae and posterior clinoid processes, which become decalcified. As the neoplasm expands, the process of demineralization involves the sellar floor and the fossa increases in depth and anteroposterior diameter, assuming the so-called "ballooned" appearance. Eventually the tuberculum sellae and anterior clinoid processes also become atrophic. In acromegaly, additional extrasellar changes are the rule. The calvarium is thickened, the paranasal sinuses enlarged and the mandible prominently overdeveloped.

In the relatively rare case of chromophobe adenoma in which the sella turcica remains normal in size, doubt may arise as to the nature of the lesion and additional information may be necessary through pneumencephalography. Films taken in the upright position to permit adequate visualization of the basal cisterns are particularly informative. Tumors arising from the pituitary body displace the cisterna interpeduncularis dorsally and either obliterate the cisterna chiasmatis entirely or else displace it rostrally. Deformities may also be noted in the hypothalamic portion of the third ventricle as well as in the floor of the frontal horns of the lateral ventricles.

The clinical manifestations of acromegaly are so striking that confusion in diagnosis is hardly likely to occur. Difficulties may arise, however, in distinguishing between a chromophobe adenoma and a number of other parasellar lesions, such as a craniopharyngioma, suprasellar meningioma, glioma of the optic chiasm, aneurysm of the circle of Willis and rarely an epidermoid, chordoma, tuberculoma or gumma. Craniopharyngiomas are primarily, though not exclusively, tumors of childhood, are frequently associated with adiposogenital dystrophy and papilledema in children and in about 80 per cent of cases show spotty calcification above the sella turcica. Gas studies are likely to reveal a deformity of the third ventricle, especially of the hypothalamic portion, obliteration of the cisterna interpeduncularis and a defect of the rostral end of the cisterna pontis. The suprasellar meningiomas occur largely in women past middle age, do not give rise to endocrine changes, are usually unaccompanied by enlargement of the sella turcica and may produce a hyperostosis overlying the tuberculum sellae. Pneumencephalography typically reveals displacement of the cephalic margin of the cisterna interpeduncularis caudally, together with a concave defect rostrally. Truncation of the frontal horns of the lateral ventricles or deformities of their inferior margins may also be evident. Gliomas of the optic chiasm and nerves give rise to symptoms usually during childhood and may be associated with manifestations of von Recklinghausen's disease. The findings on funduscopic examination are variable, as are the visual field changes. These tumors may undermine the anterior clinoid processes, thereby producing a J-shaped sella turcica. Enlargement of the optic foramina can often be demonstrated. The abnormalities observed in the pneumencephalogram consist of obliteration of the cisterna chiasmatis and a slight deformity of the cephalic border of the cisterna interpeduncularis. The diagnosis of an aneurysm of the circle of Willis is reasonably certain whenever a history of subarachnoid hemorrhage is elicited. The onset of a sudden ophthalmoplegia together with pain about the eye and forehead is also very suggestive of an aneurysm. Occasionally, the demonstration of a curvilinear streak of calcium

on the x-ray film offers a clue to its presence. Visualization by angiography will usually resolve all doubt.

The syndrome of pituitary basophilism (Cushing's syndrome) has become a well recognized clinical entity since Cushing's original description. It is a disease which affects women much more frequently than men. The manifestations of this syndrome are painful adiposity of the face, neck and trunk, kyphosis associated with osteoporosis and backache, and occasionally with compression fractures of the vertebrae, hypercalcinuria frequently with nephrolithiasis, amenorrhea in women and impotence in men, hirsutism without other evidence of virilism, hypertension, a thin, reddish skin, purplish striae, acrocyanosis, a tendency to bruise easily, purpura, diminished resistance to cutaneous infection, polycythemia, slight exophthalmos, edema of the lower extremities, weakness and hyperglycemia. According to Albright,[1] the significant laboratory findings are an unresponsiveness to an alimentary hyperglycemia, insulin resistance, and usually a moderate increase in 17— ketosteroid excretion. Recently an excessive output in the urine of 11— oxycorticosteroids has been demonstrated in patients with this condition.[20]

The underlying pathology of this disease has been succinctly epitomized by Albright:[1] "A patient with Cushing's syndrome at autopsy may or may not have a cancer or an adenoma of one adrenal cortex; one who does not have this may or may not have a basophile adenoma of the pituitary; one with a basophile adenoma of the pituitary may have hyperplasia of the adrenal cortices, and in the author's opinion, probably always would be found to have if the criteria for making the diagnosis and for determining hyperplasia were sufficiently accurate; finally, a patient who has neither a basophile adenoma nor a tumor of one adrenal cortex may have and probably always does have hyperplasia of the adrenal cortices."

Treatment. A considerable body of evidence exists to bear out the contention that whenever possible, radiation therapy is the method of choice in the treatment of pituitary tumors. This is generally conceded to be the case as far as the eosinophilic adenomas are concerned, but there still is a difference of opinion as regards the effects of roentgen therapy on the chromophobe tumors. Thus, it was only recently stated in a discussion on the radiation treatment of cerebral neoplasms that "chromophobe adenomas are no longer treated by radiotherapy for no conclusive evidence of their response to treatment has been obtained."[14]

In a monograph on pituitary tumors published in 1925, Dott and Bailey[7] stated: "To summarize the treatment of hypophyseal adenomata, we advocate a trial of x-ray therapy unless there is imminent danger of loss of vision, when a sellar decompression should be done forthwith. During the course of treatment, the progress of events should be carefully controlled by repeated perimetric examinations. If visual impairment becomes greater, a transphenoidal operation should be carried out, with partial removal of the tumor. Later, should symptoms referable to a lateral cranial extension of the tumor persist, a transfrontal operation should be undertaken. Following all these measures, x-ray treatment should be given in the hope of retarding the further growth of the tumor. They should at first be administered at intervals of three or four weeks, and later the treatment should be administered as the progress of the particular case demands."

The results of the roentgen treatment of five cases of pituitary adenomas, four of which were chromophobe tumors and one a chromophile tumor, were reported by Dyke and Gross[9] in 1931. Marked improvement of vision was achieved in three cases, while in the remaining two cases, there was no further loss of vision. In every case, headache was entirely relieved.

In 1936, Dyke and Hare[10] reviewed a series of sixty-three cases of pituitary tumor treated by irradiation. There were

twenty-five chromophile adenomas and thirty-eight chromophobe adenomas in this group. The effects on the visual fields and on visual acuity were emphasized. Of the thirty-eight chromophobe adenomas, 26.4 per cent showed moderate to marked improvement, 26.3 per cent were unchanged and 47.3 per cent became worse. Of the twenty-five acromegalics, 76 per cent showed an interruption in the progress of the disease or definite improvement; 24 per cent showed progression of symptoms despite treatment. Henderson[13] compared the data of Dyke and Hare pertaining to the chromophobe tumors with the statistics from Cushing's material. He pointed out that following operation, 68 per cent of the cases improved and 5.4 per cent became worse, whereas irradiation resulted in improvement in only 26.4 per cent, while 47.3 per cent continued to become worse. Dyke and Davidoff[8] subsequently indicated that Henderson's comparison was not altogether valid. The figures quoted by Henderson represented immediate results following operation, whereas the data submitted by Dyke and Hare represented late results, on the average of four years after therapy. It was further indicated that according to Henderson's figures, about 50 per cent of all patients operated upon showed evidence of a recurrence of the tumor. Seventy per cent of the recurrences appeared within two years and 95 per cent within five years of operation. The percentage of recurrences in Cushing's series, as reported by Henderson, was much less in those patients who received postoperative irradiation.

Sosman[19] (1937) expressed an opinion to the effect that following irradiation, "satisfactory remission of symptoms and signs will be obtained in over 50 per cent of patients with chromophobe adenomas, in 90 per cent of chromophil adenomas, and in the majority of basophil adenomas." The technic employed by Sosman in the treatment of pituitary tumors is very similar to that of Dyke and Davidoff.

Very favorable results in the treatment of twenty-five cases of pituitary tumor by irradiation were reported by Kerr and Cooper[16] in 1942. All but two cases were chromophobe adenomas. The results were divided into "excellent," "good" and "poor" depending upon whether there was (1) an increase in visual fields, loss of headache and general improvement in the patient's over-all condition, (2) arrest of the process with loss of headache and (3) no demonstrable good effects of the treatment. Excellent results were obtained in 56 per cent, good results in 20 per cent and poor results in 24 per cent. Kerr[15] has subsequently reported the results of a follow-up survey of these twenty-five patients and expanded the series by adding another twenty-six cases. Of the original twenty-five cases, one was proved not to have a pituitary tumor, thereby reducing the series to twenty-four cases. The results were excellent in 58.3 per cent, good in 20.8 per cent and poor in 20.8 per cent. Of the entire series of fifty cases, 58 per cent had excellent results, 12 per cent good results and 30 per cent poor results. The group was composed of thirty-seven chromophobe and eleven chromophile adenomas, one mixed tumor and one basophilic adenoma. Seventy-two per cent of the chromophile cases and 70 per cent of the chromophobes responded favorably to treatment. The single case of basophilism expired suddenly one week after the completion of therapy. It would appear from this study that favorable results which last for a period of six years are usually of a permanent nature. Kerr advises that all types of pituitary tumor be initially treated by irradiation. Continued decrease in the size of the visual fields two months after the cessation of treatment is considered to be an indication for operation. Kerr treats his patients with a large amount of radiation administered in one course. Four portals are utilized, two temporal, a frontal and a vertical. The physical factors are 200 KV, 1.95 mm. Cu HVL, 50 cm. distance and a 5 cm. cone. One hundred roentgens are given to one field the first day. If there is no reaction, two fields receive 100 r each the second day

and thereafter 200 r are administered to each of two fields per day. A total of 2,000 r measured in air is delivered to each field.

Of the fifty-nine cases of chromophobe adenoma treated with irradiation in this series, improvement was obtained in seventeen. Still another patient who showed regression five years after radiation therapy and improved again following further roentgen treatment raises the figure of cases improved to eighteen or 30.5 per cent of the total treated. Visual failure was halted in an additional ten patients, three of whom were operated upon nevertheless. The effect of radiation on the remaining seven patients can be justifiably considered beneficial since their vision was stabilized at a satisfactory level. Thus a total of twenty-five patients or 42.4 per cent were favorably affected by radiation therapy. Twenty-nine chromophile adenomas were treated with irradiation. Improvement resulted in eighteen or 62 per cent.

The accumulated evidence would appear to justify the practice of initially treating pituitary tumors with irradiation unless vision is dangerously close to amaurosis. The primary purpose of operating on a pituitary adenoma is not to remove the tumor in its entirety but to relieve pressure on the optic chiasm. It is technically impossible to remove the tumor completely; and even if total removal were feasible, it would be accomplished at the expense of complete destruction of the pituitary gland. That relief of pressure on the optic chiasm can be achieved by irradiation alone in an appreciable percentage of cases has been adequately demonstrated. The factor of operative mortality also bolsters the argument in favor of irradiation. Exclusive of recurrences, operation was performed in fifty-two cases of chromophobe adenoma, in five cases of chromophile adenoma and in one case of a pituitary malignancy. Operation was also carried out in a patient with Cushing's syndrome for the purpose of implanting radon seeds into the pituitary

gland. There were eight postoperative deaths with a resultant mortality figure of 13.6 per cent. This operative mortality is perhaps higher than that reported from some other clinics and distinctly higher than that in Dr. Cushing's series. One thing must be emphasized and that is that Dr. Cushing, who did not believe in irradiation, selected for operation all cases in which a pituitary tumor was suspected, the earlier in the disease the better. According to our attitude, however, the earlier cases with fewer symptoms, better physical status and lesser degree of depression of pituitary function are selected for radiation therapy. Operation is reserved for the cases in the latter group which fail to respond to irradiation. Otherwise surgical treatment is applied to late cases with advanced local, as well as endocrinologic disturbances, extensions of the tumor into the intracranial cavity and bizarre primary locations of the tumor outside the pituitary fossa. Such cases are obviously greater surgical risks and would naturally carry a higher mortality.

Failure to achieve improvement with irradiation cannot be attributed to cystic changes within the tumor alone. Of twenty-three cases of chromophobe adenoma unimproved or worse following roentgen therapy and operated upon, cystic tumors were found in only five. Variations in the degree of sensitivity to the effects of roentgen rays are probably a more common cause for the refractory behavior of many tumors.

Twenty-eight patients with chromophobe adenomas were subjected to surgery following radiation therapy. Operation followed by roentgen therapy in all but six cases (four postoperative deaths) was performed in another twenty-three patients. Of a total of fifty-one patients operated upon, thirty-four were improved. Four patients were unimproved, in each case vision prior to operation having been exceedingly poor. There were nine instances of recurrence. The treatment of recurrences followed the same plan advocated for cases receiving initial therapy.

Five patients with acromegaly were treated surgically. Improvement resulted in two of them. Two others continued to complain of headaches. The remaining patient was operated upon only recently.

Since the syndrome of pituitary basophilism may be associated with an adrenal tumor, diagnostic procedures to investigate this possibility must be instituted. Pyelography and perirenal air insufflation are routine procedures and even if they should provide negative evidence, exploration may still be advisable. Having excluded an adrenal tumor, irradiation to the pituitary may be attempted. The evidence in favor of this form of therapy is not especially convincing. No definitely beneficial results were noted in the three cases recorded by Dyke and Davidoff.[8] Eisenhardt and Thompson[11] reported temporary improvement in only one of eighteen patients treated with irradiation. Favorable results have been reported by Cushing[5] and Sosman[19] and recently by Ellman and Vilvandré[12] and by Luft.[17] No obvious improvement was noted in the three cases in the authors' series. On the assumption that a state of hyperpituitarism exists in Cushing's syndrome, Pattison and Swan[18] implanted radon seeds into the sella turcica of two patients with this disease who had been treated ineffectually with radiotherapy. Considerable improvement resulted though it was obvious that the disease had not been cured. This form of treatment was employed in one case in the present series. Insufficient time has elapsed to evaluate the result in unequivocal terms, but some clinical improvement at least appears to have been achieved thusfar. Albright[1,2] has suggested that testosterone and potassium chloride may be valuable adjuncts in the treatment of this condition.

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LATERAL RUPTURE OF CERVICAL INTERVERTEBRAL DISCS

INCIDENCE AND CLINICAL VARIETIES

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LATERAL rupture of the intervertebral discs in the cervical spine is common. Symptoms are, as a rule, mild and do not require surgical intervention; total disability from this lesion is seldom encountered. The actual frequency of ruptures in this region is difficult to determine.

An idea of their relative incidence, however, may be gained from several sources; the x-ray department of a general hospital supplies considerable data. During a six months' period (January to June, 1947) a total of 5,557 patients was examined in the x-ray department of the Baptist Memorial Hospital This group included patients from the hospital, from the outpatient department and from the adjoining medical office building. In sixty-nine patients, or 1.2 per cent, x-rays showed changes characteristic of a ruptured cervical disc. In a number of instances, x-rays were requested of the shoulder and teeth; the clinical data furnished with the request indicated pain in the shoulder and arm, associated with a stiff and painful neck. Additional films were taken of the cervical spine; the source of trouble in most instances was found to be in the discs.

Data was obtained from a second source, a medical clinic (Lyle Motley). In 758 patients (March to September, 1947) a diagnosis of ruptured cervical intervertebral disc was made in thirty-three instances, or 4.3 per cent. While the diagnosis was not confirmed by surgery or postmortem examination in any patient, the classical x-ray and clinical findings were present in all.

Such a high percentage in this group is surprising. These were patients referred for diagnostic study, many of them suffering with pain in the precordium, supposedly of cardiac origin.

In the practice of neurosurgery one would expect this condition to be encountered more frequently. From our own records during the past six months (January through June, 1947) a diagnosis of ruptured cervical disc was made forty-nine times, or 1.9 per cent of 2,637 patients. All of the patients from this source who complained of pain in the occiput, neck, shoulder, arm and fingers were asked the routine questions and a routine examination was made for ruptured cervical disc. If the clinical picture was compatible with this condition, routine x-ray examination of the cervical spine was made. The diagnosis was verified at operation in eight patients in whom the severity and duration of the symptoms and the failure of conservative measures led to the recommendation and acceptance of surgical treatment. In the other forty-one patients, the diagnosis was based upon a characteristic history, typical findings, x-ray evidence and therapeutic tests. There were no deaths, consequently the diagnosis was not confirmed by postmorten examination in any of the patients.

The certainty of the diagnosis in some of these patients may be questioned but, on the other hand, in spite of the interest in these lesions in each organization, some cervical disc ruptures probably were overlooked. At any rate, there can be little doubt that rupture of cervical discs is responsible for a large number of occipital headaches, stiff and painful necks, shoulder and arm pains extending into the hands and fingers and anginalike pain. These statistics, although covering a short period and obtained from only three sources, indicate that this condition is sufficiently common to play an important part in diagnostic

roentgenology and diagnostic medicine as well as in orthopedic surgery and neuro-surgery.

There are two groups which are clinically and pathologically different; the acute variety is characterized by sudden onset following definite trauma such as jerking of the neck. The pain is intolerable, the neck is stiff and nerve root involvement is evident from the sensory and reflex changes. Even weakness of the muscles and vasomotor disturbances are not infrequent. X-ray findings are limited to loss or reversal of the normal curve since the amount of displaced cartilage is not sufficient to result in narrowing of the interspace, nor has sufficient time elapsed for degenerative changes or bone reaction. In this group relief is not obtained by rest or traction and early operation is demanded by the excruciating pain. Operative findings in patients exhibiting this clinical picture are flattening and compression of the nerve root. The root is not adherent but is exquisitely tender; a small nodule of fibrocartilage bulges beneath the stretched and thinned posterior ligament in the manner usually found in the lumbar region. Following removal of the small mass of fibrocartilage, the nerve returns to its rounded contour. Relief is immediate and dramatic and the function of the neck and affected nerve is usually restored promptly.

The second group is the chronic type following mild or repeated trauma; the patient may not have any recollection of injury. At first there are cricks in the neck, then pain in the occiput, shoulder, arm, fingers and precordial region and numbness and tingling of the fingers. It is in this group that neuritis, tendonitis, bursitis, myositis, fibrositis, angina and the scalenus anticus syndrome are usually thought to be responsible for the symptoms. These patients are able to raise the affected arm over the head without discomfort. The pain is increased by downward pressure on the head and flexion of the neck and is relieved by traction on the head. Tenderness is elicitied by pressure on the rhomboid and the subclavicular regions and over the affected nerve trunk in the neck. Sensory and reflex changes may be present and atrophy of the affected muscles evident. In this group x-rays show, in addition to the loss or reversal of curve, narrowing of the affected interspace, or interspaces and osteophytes; the latter can usually be demonstrated in oblique views, extending into the neural foramen laterally so as to impinge upon the nerve root.

In the chronic group, surgery is indicated when severe pain and disability of long duration or of frequent recurrence has failed to respond to conservative measures such as bed rest and head traction, followed by the wearing of a suitable collar or support. The operative findings are very different from the acute variety. The nerve root is found to be flattened, adherent and compressed by a ledge of hard material narrowing the neural foramen laterally. The nerve root is squeezed so tightly in its foramen that even a small probe can be passed beside it with difficulty, if at all. The foramen is never foreshortened sufficiently in its longitudinal diameter to compress the nerve root, regardless of the degree of narrowing of the interspace. This may readily be proven by examining a skeleton with no padding whatever between the vertebral bodies.

In recent cases, the nerve root can be displaced either upward or downward with reasonable ease and the nodule of cartilage removed. In the chronic variety, it is more difficult to displace the nerve; removal of the hard material cannot be accomplished without risk of damaging the nerve. In some instances the calcified protrusions are sharp; excision with a chisel, curet or dental bur is indicated. Ordinarily, nerve root compression can be relieved by enlarging the neural foramen laterally at the expense of the facets.

Neither of these types seems to be related to the "explosive" central ruptures found in patients injured in diving accidents and violent automobile accidents; these were formerly attributed to "subluxation." Nei-

ther are they apparently related to the central ruptures which protrude slowly, simulating spinal cord tumors.

Lateral rupture of a cervical intervertebral disc, although very common and the cause of prolonged and recurrent symptoms, seldom causes incapacity. Surprisingly few of these ruptures are severe enough to require surgery for relief; most of these are of the acute variety with soft ruptures. Doubtless, many of the milder syndromes, with only cricks and stiffness in the neck and transient radiating pain, recover spontaneously without recognition.
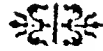
In neurosurgery, as in other fields, the most important diagnostic factors are the careful history and physical examination. Valuable adjuncts, such as perimetry, audiometry, roentgenography, electroencephalography, and cerebrospinal-fluid studies, may be essential.

RUPTURED INTERVERTEBRAL DISCS IN THE LOWER LUMBAR REGIONS*

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THE disc controversy continues un-abated fourteen years after the classic description of the disorder by Mixter and Barr.[1] Unfortunately, the controversy is not confined to treatment methods but includes pathologic and clinical diagnostic problems as well.

In Mixter and Barr's original article this disorder was named "rupture of the inter-vertebral disc with involvement of the spinal canal." In a later paper Mixter and Ayer used the term "herniation or rupture of the intervertebral disc into the spinal canal."[2] These terminologies were an attempt to state accurately the exact anatomic and pathologic conditions found in the diseased discs. In 1934 Peet and Echols called the disorder "herniation of the nucleus pulposus."[3] Later, the term "protruded intervertebral disc"[4] was introduced into the literature. This term had broader implications than the ones of Mixter et al., for it included those discs that protruded backward into the spinal canal without frank rupture of the annulus fibrosus.

Dandy[5] proposed a new terminology—a "hidden" or "concealed" disc. His description applied more to a condition found at the operating table than to an actual pathologic state demonstrated on post-mortem material. Later he introduced an entirely new conception into disc surgery, i.e., a large percentage of patients suffering from disc symptoms have multiple ruptured discs.[6]

In the beginning the neurosurgeons pioneered the problem; soon the orthopedic surgeons became interested in it. Their points of view were usually quite dissimilar to those of the neurosurgeons, for they considered a spinal fusion operation of one type or another to be a paramount con-sideration. More recently the general surgeons, particularly the industrial sur-geons, have joined the ranks of disc en-thusiasts. At present the patient with low back and leg pain is apt to receive about as many opinions as doctors whom he has consulted about his complaints. It is small wonder that reports of distressingly bad results[7] are creeping into the literature.

Our purposes in this report are: (1) To restate and clarify the fundamentals of the problem, i.e., anatomic, physiologic and pathologic features; (2) to re-evaluate the accuracy of the neurologic symptoms and signs; (3) to clarify the treatment methods based upon twelve years of active interest and experience and (4) to present a sample follow-up study of our results.

Anatomic Considerations. A thorough knowledge of the anatomic relations in the lower lumbar portion of the spine is essen-tial to a clear understanding of the symp-toms and signs of ruptured intervertebral discs.

Each intervertebral disc is composed of three parts: (1) annulus fibrosus, (2) nucleus pulposus, and (3) cartilaginous plates. (Fig. 1.)

The *annulus fibrosus* forms the periphery of the disc and is composed of concentric layers of fibrous and elastic tissue. Ante-riorly, the annulus is thick and well defined and its fibers are blended with the strong anterior longitudinal ligament. Posteriorly, the annulus is about one-half the thickness of that on the anterior surface and is separated from the spinal canal by the
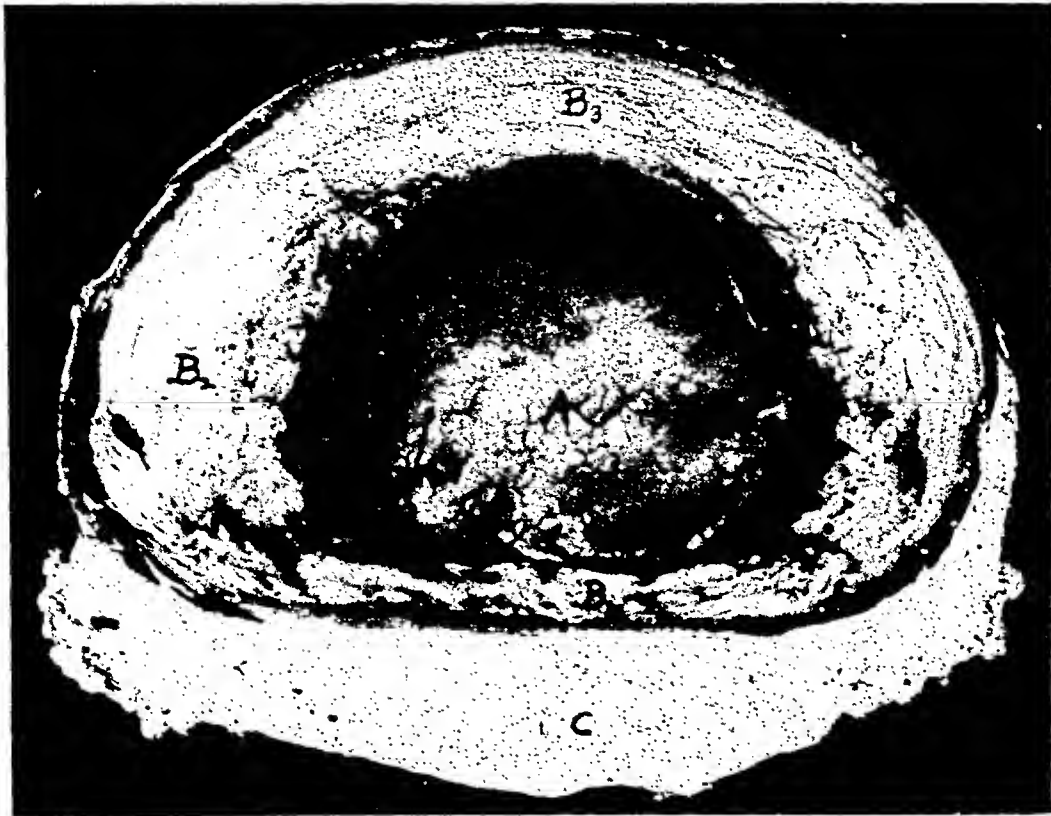
Fig. 1. Intervertebral disc dissection. The fourth and fifth lumbar vertebrae with intervening disc were removed intact from a fresh autopsy specimen. The intervertebral disc was split by cutting through the mid-portion of the annulus fibrosus, thus leaving two halves of the disc with smooth surfaces. The nucleus pulposus was carefully dissected from each half and the two parts glued together. Then the whole nucleus was covered with modeling wax. The nucleus and the depression from which half of it had been dissected were painted yellow. The remainder of the disc and the fifth lumbar vertebra were painted white. The nucleus was placed in its bed and the specimen photographed on panchromatic film. A, nucleus pulposus; B₁, B₂ and B₃, annulus fibrosus; c, body of the fifth vertebra.

posterior longitudinal ligament which is thin and weak, giving no support to the annulus except in the midline where it becomes somewhat thickened.

The *nucleus pulposus* occupies the approximate center of the disc. The point at which the annulus ends and the nucleus pulposus begins is not sharply defined. However, the main nuclear mass is quite distinct grossly from the remaining disc material. The nucleus pulposus is soft and has a semi-fluid consistency, particularly during the first three decades of life. Its water content is gradually reduced with age, until in the fourth and fifth decades it loses much of its elasticity. Histologically, the nucleus pulposus is composed of loose fibrous tissue interspersed with cartilage cells.

The *cartilaginous plates* separate the annulus and the nucleus from the adjacent vertebrae. The layer of cartilage is well defined in its central portion where it protects the adjacent vertebrae but thins toward the periphery where it blends with the annulus fibrosus.

Until recent years it was believed that there were no sensory nerves in the posterior annulus fibrosus. It has now been amply demonstrated that the annulus fibrosus is richly supplied with sensory nerves.[5] This anatomic fact confirms the clinical observations that manipulation of the annulus fibrosus in patients operated upon under local anesthesia causes severe low back pain.

The relation of the fifth lumbar nerve to the disc between the bodies of the fourth and fifth lumbar vertebrae and the relation of the first sacral nerve to the lumbo-

sacral disc are especially important, since over 98 per cent of all ruptured lumbar discs occur at these levels.

The spinal cord terminates opposite the intervertebral disc below the first lumbar vertebra. The nerve roots of the cauda

FIG. 2. Diagrammatic representation of the thecal sac and the lower lumbar and sacral nerves to the pedicles and intervertebral discs of the lower part of the spine.

equina are freely mobile in the large lumbar canal except as they approach their points of exit where they become fixed. (Fig. 2.) It is, therefore, apparent that only those roots that are compressed near their points of exit cause symptoms and signs of localizing value unless the lesion is a tremendously large one which uniformly compresses the roots of the cauda equina. At the level of the fourth lumbar disc the fifth lumbar nerve is contained in the subarachnoid space, fixed laterally against the dura one vertebra higher than its exit. The dural sleeve of the fifth lumbar nerve emerges below the fourth lumbar disc and passes downward beneath the pedicle of the fifth

lumbar vertebra through its intervertebral foramen. (Fig. 3.) This relation holds between all the lumbar nerves and their respective discs. In contrast, the dural sleeve of the first sacral nerve separates from the thecal sac above the lumbosacral
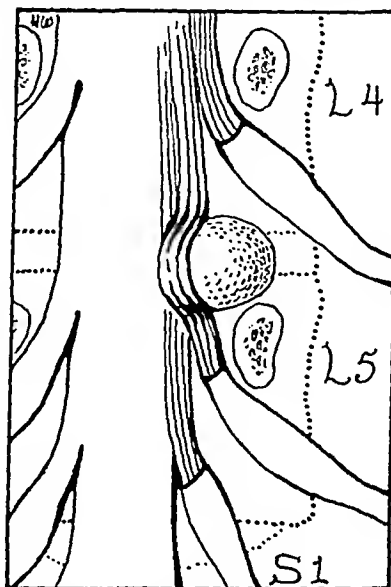
FIG. 3. Diagram illustrating the mechanism of compression of the roots of the fifth lumbar and first sacral nerves from a lateral herniation of the nucleus pulposus at the fourth lumbar interspace.

disc and can thus be compressed without deforming the sac.

It is necessary that any physician who wishes to localize a ruptured intervertebral disc on the basis of clinical findings has a thorough anatomic knowledge of the distribution of the fourth and fifth lumbar roots and the first sacral root. The sensory dermatomes of the fourth and fifth lumbar and the first sacral nerve roots are complex and a thorough knowledge of their complexity need not be discussed at this time because only certain portions of the dermatomes are essential for accurate diagnosis. As an example, the patch of skin on the medial aspect of the foot and leg just above the ankle represents the distal portion of the sensory dermatome of the fourth lumbar nerve, whereas, the area of skin on the dorsal surface of the great toe and a band · extending directly upward on the

foot from the great toe is the distal distribution of the fifth lumbar dermatome. The first sacral dermatome supplies the skin on the lateral aspect of the foot, the outer two toes and perhaps the third toe as well. The dermatome extends over the

In lesions affecting the fourth lumbar nerve (L3 disc) the quadriceps femoris is most affected. With involvement of the fifth lumbar nerve (L4 disc) the anterior tibial group particularly the extensor of the great toe may show evidence of weak-
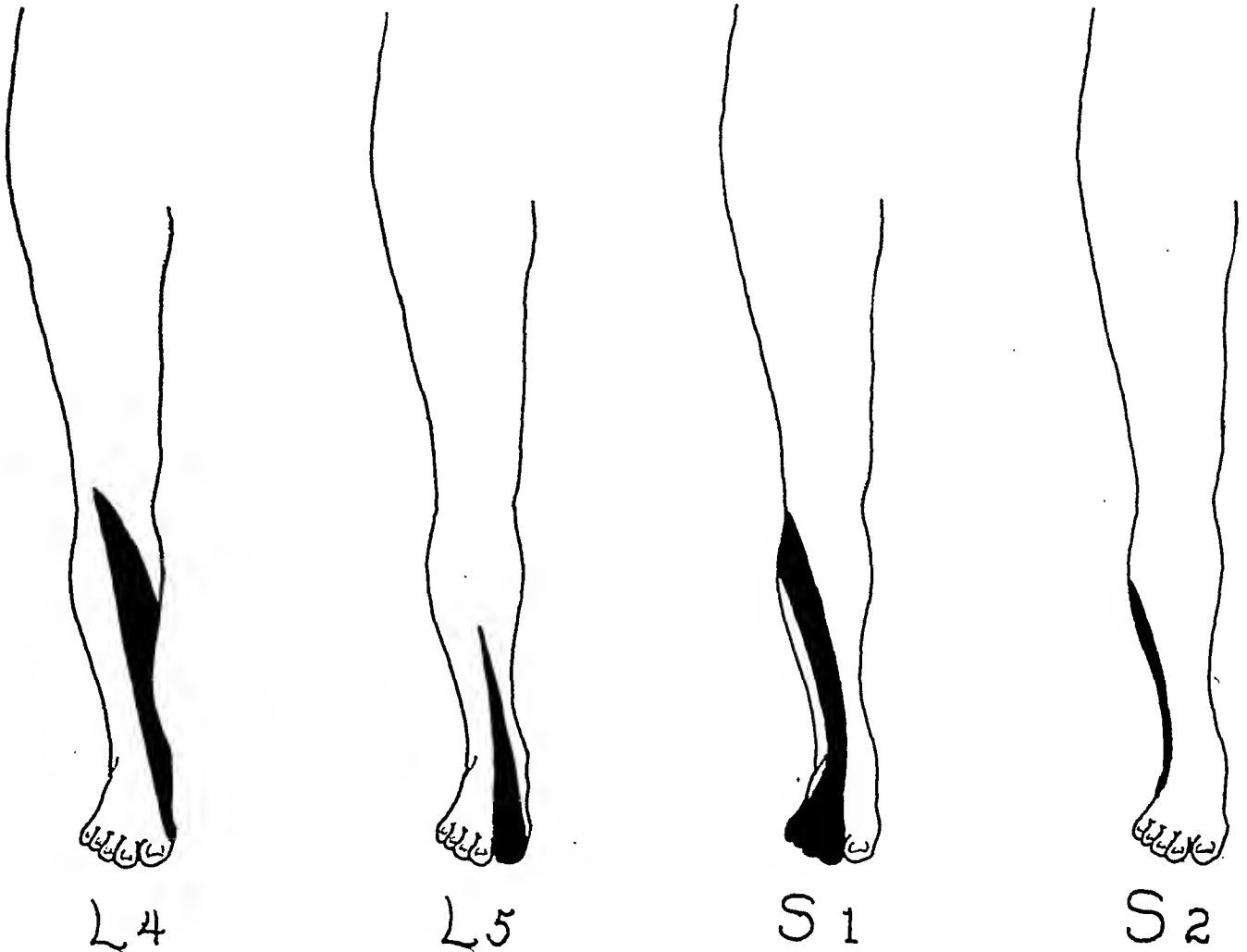
FIG. 4. Distal sensory distribution of L4, L5, S1 and S2 nerves.

lateral aspect of the ankle up the lateral aspect of the leg to just below the knee. (Fig. 4.)

The motor innervation of the two lower lumbar and first sacral nerves has considerable importance in diagnosis and localization. However, all of the muscles of the lower extremity have a dual or even a multiple segmental innervation so that paralysis of a muscle or muscle group is seldom seen in disc lesions. In the great majority of disc lesions only a single root is affected; hence the muscle or muscular group supplied by it would show only a minor degree of weakness.

ness. In lesions of the first sacral nerve (L5 disc) the posterior calf muscles (gastrocnemius and soleus) are most often affected and weakness in these muscles may be easily demonstrated by having the patient walk on his toes. If weakness is present, the heel of the affected side will drop to the floor or at least show a tendency to do so.

The tendon reflexes are of great importance in diagnosis. If the knee jerk is depressed, it points definitely to involvement of the fourth lumbar nerve, i.e., L3 disc. An absent ankle jerk indicates a lesion of the first or first and second sacral nerves, i.e. L5 disc.

There are no very reliable reflex changes associated with a fourth lumbar disc lesion. As a matter of fact, given a patient with a characteristic history and with physical findings of a ruptured disc, who has normal knee and ankle jerks, the presumptive evidence is that the lesion is at the fourth lumbar disc. The posterior tibial muscle receives a large portion of its motor innervation from the fifth lumbar nerve and its tendon reflex may be abolished with lesions at this level. However, we have found this reflex difficult to elicit consistently and we doubt its practical value in diagnosis.

Physiologic and Pathologic Considerations. The intervertebral discs have frequently been compared functionally to a hydraulic shock absorber. Certainly, the structure of the intact disc would indicate that such a comparison is justified.

The misstatement is frequently made that the nucleus pulposus is highly expansile. Actually, the nucleus pulposus is incompressible and due to its plastic nature obeys the laws of fluids. It is the tension of the annulus fibrosus which keeps the nucleus pulposus under pressure, even when the intervertebral disc is not bearing weight.

The annulus fibrosus can be compared to a strong but somewhat elastic membrane firmly binding the vertebral bodies together. The nucleus pulposus which fills the cavity separating the vertebral bodies can be compared to a fluid. Although the nucleus pulposus is plastic rather than fluid, it behaves as a fluid in response to the limited degree of displacement to which it is subjected in movements of the vertebral column.[9] Barr's experiments are most illuminating in this regard.[10] He found that by compressing the vertebral column in a vice he could produce marked distention of the posterior annulus fibrosus. When this pressure was released, the annulus fibrosus returned to a normal shape. However, if he pierced the annulus fibrosus with a needle while the disc was compressed, the annulus fibrosus ruptured where it was penetrated, allowing the nucleus pulposus to extrude itself outward. When this occurred, the intervertebral disc lost its elasticity and failed to return to its former state.

When injury to the posterior annulus fibrosus or when the laminae of hyalin cartilage has allowed the nucleus pulposus to escape, the annulus fibrosus is subjected to forces which are alien to it. It becomes then merely a washer resisting direct force. With the fluid medium for even distribution of the transmitted force gone, movements are forced to bear upon a small part of the intervertebral disc at any given time. The disc is then in a position to be ground between the adjacent vertebral bodies. If the vertebral body is weakened by disease, it, too, may be ground away; at least it will become sclerosed and probably produce spurs to resist the unusual and poorly distributed force to which it has been subjected.

It is obvious, therefore, that when the anatomy of the disc is altered, repair by the usual processes may occur; but these reparative processes cannot hope to restore the normal physiology of the disc.

Carrying this reasoning into clinical application, it may be stated unequivocally that once a disc is ruptured *no surgical effort can hope to restore its anatomic integrity.* The natural healing processes may close the tear in the annulus fibrosus, or may even cause fibrous union of the contiguous cartilaginous plates, but nothing remotely resembling the normal shock-absorbtive mechanism can be restored. This reasoning applies with equal force to the disc that has been ruptured accidentally as well as to the normal disc that the surgeon who, through lack of knowledge or carelessness, has opened at the operating table in an attempt to find the diseased one.

We do not pretend to know how many different types of disc disorders may enter into the painful back syndrome. We do know that the posterior longitudinal ligament and the posterior annulus fibrosus are richly supplied with sensory nerve endings.

We know that manipulation of these structures in a conscious patient at the operating table produces the characteristic type of low back pain usually called "lumbago." But further than this we have no exact knowledge on this subject.

We are, however, thoroughly convinced that we know the type of disc disturbance which is responsible for *unilateral sciatic pain* associated with low back disability. The lesion that produces these disabilities is primarily one of the *posterior annulus fibrosus*. As we have previously indicated, there is a constant bulging of the posterior annulus fibrosus under conditions of stress and strain. However, in the intact disc this bulge disappears immediately once stress is removed, and the annulus fibrosus again assumes its normal contour. But when the annulus fibrosus is torn, the normal pressure relationships within the disc are immediately changed. If the tear in the annulus fibrosus involves the entire structure, there may be immediate herniation of the nucleus pulposus through the tear, thus producing a bulging mass within the spinal canal. The nucleus pulposus may even break through the flimsy posterior longitudinal ligament and lie loose within the spinal canal. A hernia thus produced is obviously an irreducible one; and no matter how much conservative treatment is used, such a herniation will not be relieved by natural healing processes. The patient, however, may eventually be relieved of his symptoms by total destruction of the nerve root.

The tear in the annulus fibrosus, however, does not necessarily have to be a complete one. The tear may involve only the inner group of fibers of the posterior annulus fibrosus and, therefore, create nothing but a weakened spot in the structure. However, as one would expect with any structure in which hydrodynamic forces are called into play, that spot being the weakest will bulge when strain is applied; in fact, it will bear the brunt of all changes in pressure relationships due to motion. (Fig. 5.)

The first type of disc disorder just described is the one which is so easily demonstrated at the operating table. The second type is quite a different matter, for here one may see only a localized bulge in the annulus fibrosus, or for that matter in certain positions in the recumbent position a bulge may not even be demonstrated. However, if one palpates the disc carefully with a blunt instrument the weakened spot can usually be identified. It may occur in any position from the midline to the most lateral limit of the posterior annulus fibrosus.

The first type of disc condition (frank rupture with herniation of the nucleus pulposus) described is the type that is refractory to conservative treatment. The second type (with incomplete rupture) may be more favorable for conservative therapy. We are quite convinced that many cases of the latter type recover spontaneously or with ordinary conservative treatment if this is instituted during the first attack of pain.

Clinical Considerations. In June, 1939, one of us (R. G .S.) in collaboration with Bradford[11] suggested a group of criteria that could be relied upon in the clinical diagnosis of ruptured intervertebral disc at either the fourth or the fifth lumbar interspace. These criteria were based upon a series of ten cases in which surgical procedures were done without the aid of contrast myelography.

In March, 1940,[12] we made a report upon the clinical diagnosis of intervertebral disc lesions in the lower lumbar region based upon experience with 125 consecutive operations. We found that the criteria originally presented by Spurling and Bradford are, on the whole, accurate. In addition, we presented the localizing features of ruptured intervertebral disc at the level of the third interspace.

Now, after seven years of additional experience we shall attempt to select the most important features of the subjective and objective examination in making the
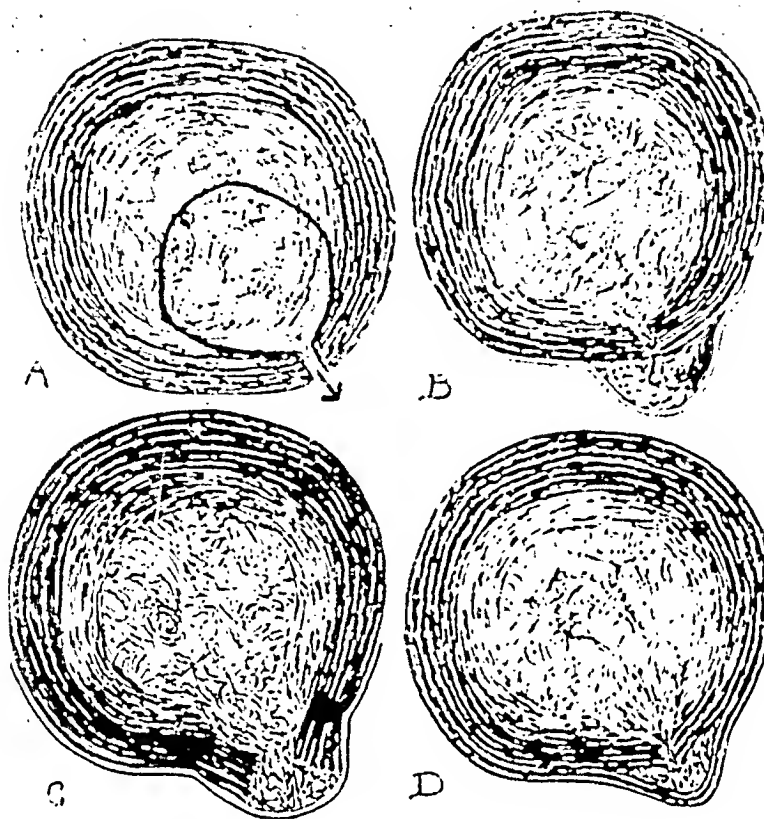
FIG. 5. The intervertebral disc is diagrammatically represented in cross section. The outer lamellae of the annulus fibrosus are represented as dense parallel bundles, the inner lamellae as lighter bundles and the nucleus pulposus as interlacing fibers. A, a line has been drawn around that portion of nucleus pulposus and inner lamellae of the annulus fibrosus which will rupture through the defect in the outer lamellae of the annulus fibrosus. B, the herniation is completed. C, a rarer type of herniation in which the outer annulus fibrosus is fractured. D, an interlamellar herniation in which the two outermost lamellae of the annulus fibrosus have distended over the mass without rupturing.

clinical diagnosis of ruptured intervertebral disc and also in accurately localizing it.

HISTORY

With rare exceptions, the first symptom of a ruptured intervertebral disc is backache. Direct trauma to the spine is not a factor in more than 40 per cent of the cases. A frequent story is that when in a bent forward position, lifting a heavy object, the patient felt a sudden catch in the back and straightening upright could be accomplished only with excruciating pain. The outstanding characteristic of the backache is its mechanical nature. It is intensified by any movement, particularly on bending forward or straightening upright. Twisting motions, such as turning over in bed, are especially painful. Relief of the pain is usually accomplished by lying perfectly still, although some patients cannot tolerate the supine position and can find comfort only in a sitting or even a jackknife position. The pain is usually intensified by coughing, straining or sneezing.

There may be one or more attacks of backache before the onset of sciatic pain. However, when trauma produces gross injury to the intervertebral disc, leg pain may occur simultaneously with low back disability. Once leg pain has started it usually follows the entire course of the sciatic nerve. The points of maximum intensity are the gluteal region, the posterior

thigh and the calf of the leg. Usually the sciatic pain remains unilateral, but occasionally it may be bilateral, and in a few cases the pain will shift from one leg to the other during the same or successive episode.

Some patients will voluntarily report

unequivocally. When doubtful the test should be considered negative.

1. Most patients with a ruptured intervertebral disc have a straight lumbar spine with obliteration or even reversal of the normal lumbar lordosis. Most of them, in
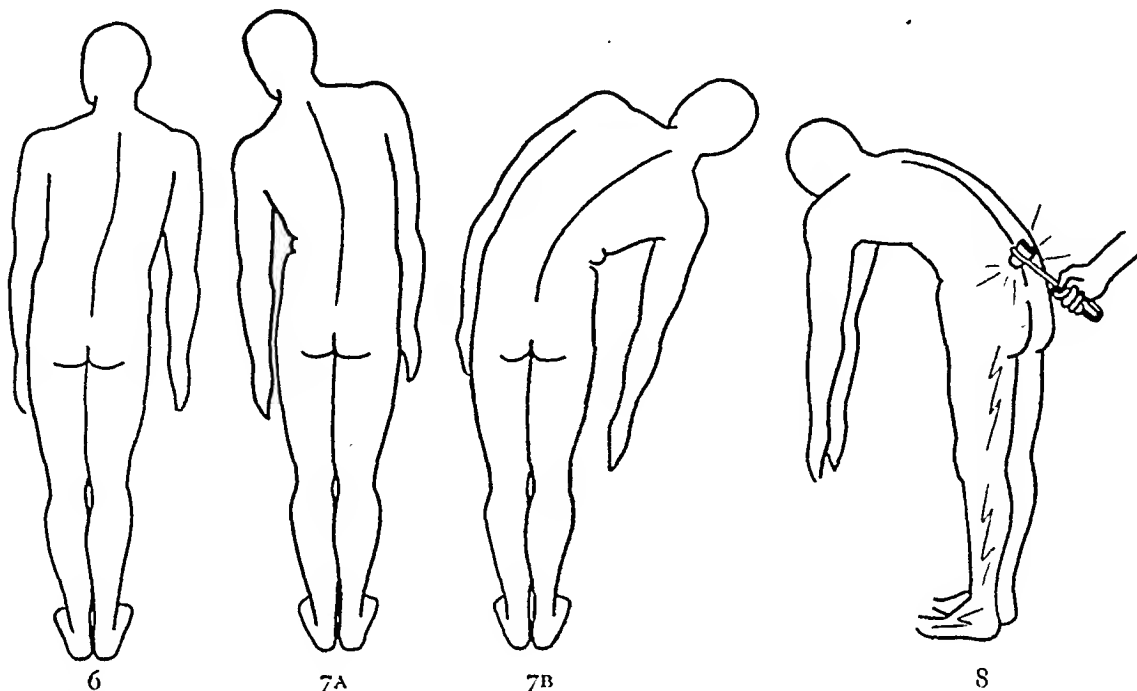
FIG. 6. Typical list from lumbar disc lesion (left).
FIG. 7. A, restricted lateral bending toward painful side; B, unrestricted lateral bending toward normal side.
FIG. 8. Production of radicular pain by deep percussion.

that certain parts of their leg are numb and that there is a feeling of "needles and pins" in certain skin segments of the leg and foot.

Partial or complete remission of symptoms is the rule when the history covers a period of several months or years. Recurrent attacks with freedom from pain between the attacks is the usual history.

Many patients report relief of symptoms by manipulative therapy. Others apparently are made worse by such procedures and obtain no relief until they have remained in bed for a few days.

EXAMINATION

The following tests have been selected as being the most useful ones in the diagnosis and the localization of a ruptured intervertebral disc. These tests must be made carefully and the results recorded

addition, show listing away from the side of the sciatic pain. (Fig. 6.) In almost every instance the pelvis on the affected side is higher than on the unaffected side. Movements of the lumbar spine are usually limited on flexion. The erector spinae muscles may be spastic and the spasm usually is greater on the side opposite the lesion or painful leg. Lateral bending to the side of the lesion is usually more painful than when bending away from the painful side. (Fig. 7.) Occasionally, one encounters a patient with alternating scoliosis; in other words, he can change his list from one side to the other simply by bending lateralward or forward.

2. Another test which we have found to be very valuable is extension of the lumbar spine combined with lateral bending toward the painful side. Aggravation of the back and leg pain with numbness and tingling

into the distal portion of the dermatome during this maneuver is considered pathognomonic of a ruptured disc. The test is performed with the patient erect. It is important that the knees are straight and that both heels are on the floor. The
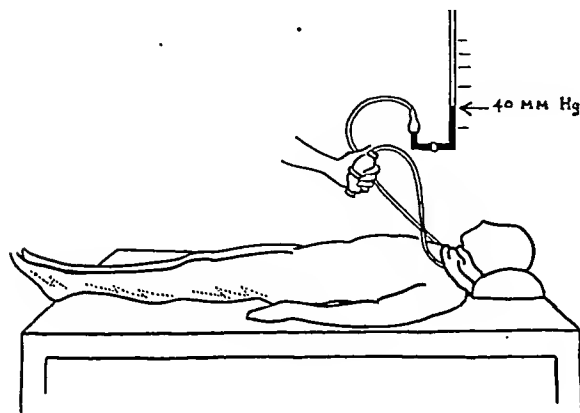
FIG. 9. Recommended method for performing jugular compression test.

examiner then grasps the patient's shoulders and slowly extends the lumbar spine and at the same time bends the patient toward the painful side.

3. *Radiating percussion pain* is also diagnostic. With the patient bent forward

calf. Radiating percussion pains thus produced are caused by waves of force transmitted through the ligamentum flavum to the affected nerve root at the site of the lesion. When positive this sign is pathognomonic of a ruptured intervertebral disc. (Fig. 8.) One may sometimes differentiate between L4 and L5 lesions by producing the radiating leg pain with the blow at one space and not at the other.

4. *The jugular compression test* is performed by impeding the venous return from the internal and external jugular veins with either digital compression or with compression by the cuff of a sphygmomanometer placed around the patient's neck. By increasing the intracranial pressure and consequently increasing the intraspinal pressure, the radicular pain in the affected leg may be aggravated. In fact, the entire pattern of the pain and even the sensory disturbances may be reproduced. (Fig. 9.)

The test may be performed with the patient either supine or upright. The venous return should be impeded until the

FIG. 10. Recommended position for straight leg-raising test.
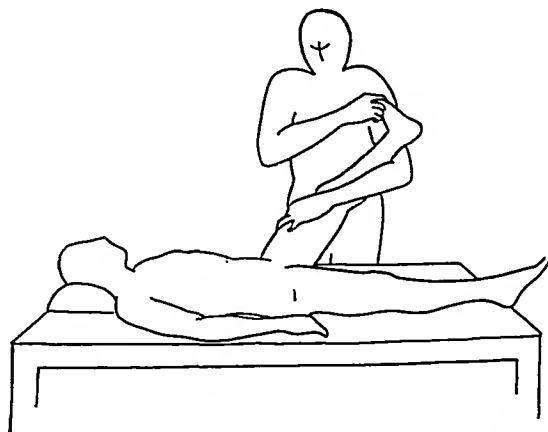
FIG. 11. Lasègue's modification of straight leg-raising test.

to the point of painful radiation into the leg a light percussion stroke over the spinous processes may accurately localize the tender point. A deeper percussion stroke to the side of the midline at this level may cause radiating pain into either the hip or down the back of the thigh or even into the

patient has a distinct feeling of fullness in his head. Many patients will state that they feel as though their eyes were "popping out." Shortly after this venous congestion has occurred, the patient whose test is positive will state that the pain in the back is worse, that it radiates into the hip,

then down the back of the thigh into the calf. He may state voluntarily that one or the other of the toes feels numb or tingles. Often by careful observation during this test the localization of the lesion may be made with accuracy. The jugular compression test when positive is pathognomonic of an intraspinal lesion involving one of the nerve roots at the lower end of the spinal canal. However, the test may be positive with cauda equina tumors as well as ruptured discs; but if other signs of ruptured disc are present and the jugular compression test is positive, it is unequivocal evidence favoring the diagnosis of this lesion.

5. *The straight leg raising test* is a valuable index of the degree of sciatic pain. (Fig. 10.) The angle at which the pain appears may also be used as a reliable index for judging the progress of the case. The Lasègue's test is a useful refinement in equivocal cases. (Fig. 11.) These two tests are almost always positive with ruptured disc in the lower lumbar region.

6. *The sensory examination* is important in diagnosis, particularly if it conforms with the other clinical findings, especially the jugular compression test. Two forms of stimuli are sufficient to demonstrate gross sensory involvement—light touch with the fingertips and painful sensation with a pin prick. In mapping out areas of sensory loss it is desirable to compare the normal with the abnormal side. In many instances, the only sign of sensory disturbance will be the patient's statement that the skin of the involved area feels thicker than normal. In questionable cases, testing with heat or cold will demonstrate sensory loss when other tests are equivocal. Paresthesias may be described as peculiar sensations, electrical shocks spreading from the point of stimulation into the remainder of the involved dermatome. They are best reproduced by lightly stroking the skin with the fingertips. For sake of emphasis it will be restated that sensory loss on the top of the great toe indicates involvement of the fifth lumbar root at the fourth lumbar

disc. Sensory loss on the lateral aspect of the foot involving particularly the lateral two or three toes indicates involvement of the first sacral root at the lumbosacral disc. Sensory loss on the medial aspect of the foot extending upward over the ankle indicates
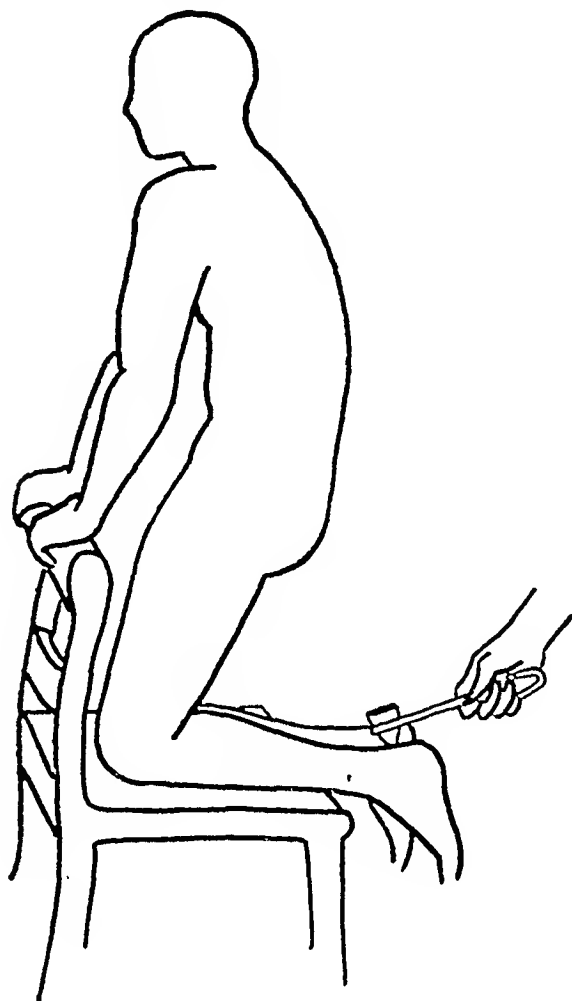
Fig. 12. Recommended position for testing Achilles reflexes.

involvement of the fourth lumbar nerve and is characteristically found in lesions at the third lumbar interspace.

7. *Reflex* changes are exceedingly valuable in diagnosis. With lesions at the third interspace involving the fourth lumbar nerve the knee jerk may be found diminished or absent. With lesions at the fourth interspace the only reflex change will be found at the posterior tibial tendon and that reflex is difficult to elicit consistently in normal subjects. Absence of the Achilles jerk is usually found with lesions at the lumbosacral disc. Since 98 per cent of ruptured discs occur at either

the fourth or the fifth lumbar interspace, the Achilles jerk is particularly important in differential diagnosis. It is a difficult reflex to elicit properly unless the examiner is thoroughly familiar with methods of producing relaxation and reinforcement of the tendon jerks. The position shown in Figure 12 is the ideal one for testing the Achilles reflex.

The reliability and accuracy of these signs have been tested upon thousands of patients by many observers. Most neurologists are in thorough agreement with their value and accuracy. If other physicians who are interested in the disc problem will study them and learn to evaluate them, the problem of the clinical diagnosis of ruptured disc will be greatly simplified.

SUMMARY OF CLINICAL SIGNS

Many ruptured lumbar intervertebral discs can be accurately localized on clinical findings alone. All cases have in common (1) low back disability and (2) pain in the distribution of the sciatic nerve. The characteristic differential points are as follows:

The Third Lumbar Interspace:

1. Tenderness to percussion lateral to the third lumbar spinous process.

2. Radiating percussion pain produced to the side of the third spinous process.

3. A positive jugular compression test with tingling appearing on the mesial aspect of the foot (the fourth lumbar dermatome).

4. The knee jerk diminished or absent; the ankle jerk normal.

5. Hypesthesia or paresthesias in the fourth lumbar dermatome.

The Fourth Lumbar Interspace:

1. Tenderness to percussion lateral to the fourth spinous process.

2. Radiating percussion pain produced to the side of the fourth spinous process.

3. Positive jugular compression test with tingling into the great toe (the fifth lumbar dermatome).

4. The knee jerk is normal and the ankle jerk is usually normal. Occasionally

the ankle jerk is diminished. The posterior tibial reflex may be absent or diminished.

5. Hypesthesia or paresthesias in the fifth lumbar dermatome.

The Fifth Lumbar Interspace:

1. Tenderness to percussion lateral to the fifth spinous process.

2. Radiating percussion pain produced to the side of the fifth spinous process.

3. Positive jugular compression test with tingling appearing on the top of the foot and the lateral three toes (sensory disturbances are always absent in the great toe).

4. Diminished or absent ankle jerk; knee jerk normal.

5. Hypesthesia or paresthesias in the first sacral dermatome.

X-RAY AND LABORATORY AIDS IN DIAGNOSIS

Plain x-ray films of the spine are often helpful in diagnosis. The most constant and characteristic feature is straightening of the lumbar spine with absence or reversal of the normal lumbar lordosis.

Narrowing of one of the lumbar interspaces is presumptive evidence which may or may not be important. The fifth lumbar interspace is normally narrower than the other lumbar interspaces. Therefore, unless there is marked narrowing with spur formation about the joint such a finding is of little or no importance.

Localized proliferative changes about the diseased joint may or may not be of clinical significance. Not infrequently, the acute ruptured disc may be above or below the level of such a localized area of arthritis.

Congenital anomalies such as spondylolisthesis may or may not be of clinical significance. Such congenital lesions may indicate an unstable spine but in our experience does not give positive or negative evidence of a probable ruptured disc at the same level or above it. We are thoroughly convinced from repeated observations that spondylolisthesis *per se* does not cause sciatic pain. If it is of severe degree (grade 4), there may be widespread disturbance of the cauda equina function below the
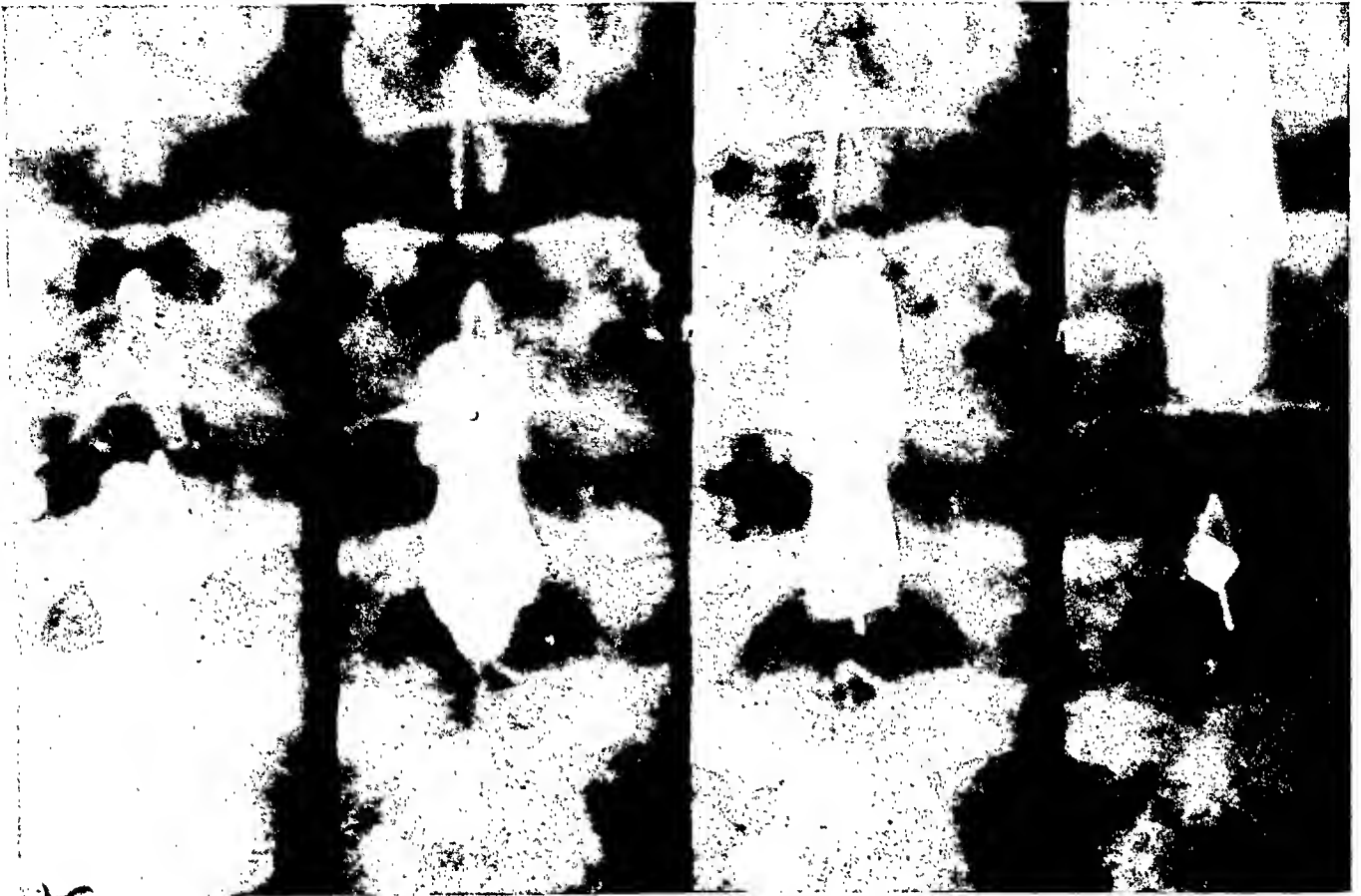
FIG. 13. Pantopaque myelogram showing normal column at L3, L4 and L5 interspaces.

level of the lesion due to obliteration of the spinal canal.

It is extremely important in studying plain films of the spine in patients with suspected ruptured intervertebral disc to observe carefully the sacroiliac joints. The Marie Strumpell type of spinal arthritis may produce symptoms strongly suggestive of ruptured intervertebral disc. Since this destructive type of arthritis usually shows up first in the sacroiliac joints, it is particularly important that the films be made in such a way that these joints may be accurately visualized.

MYELOGRAPHY

In this series of 350 verified ruptured discs, the diagnosis and localization were confirmed by myelography in 46 per cent of the cases. The remaining 54 per cent were accurately localized on clinical findings alone.

Pantopaque, an iodized oil of low viscosity, is the ideal medium for myelography. It is heavier than spinal fluid; therefore, it always seeks a dependent position in the subarachnoid space. By careful manipulation of the column under the fluoroscope it is possible to visualize accurately the subarachnoid space including the axillary pouches at any of the lumbar interspaces. If the x-ray machine is equipped with a spot film device, the operator can permanently record his fluoroscopic observations upon serial spot films. Once the examination is completed the material is usually aspirated without difficulty, leaving no trace of iodized oil in the subarachnoid space. Should a few drops of the oil be inadvertently left, it is slowly absorbed over a period of from three to four months and apparently produces no clinical symptoms of consequence. Pantopaque myelography is no more accurate than the skill with which it is done. In the hands of experts we are convinced that it is fully as accurate as the diagnosis of peptic ulcer with barium. However, there are many tricks to the examination and before any one attempts it he should be thoroughly

FIG. 14. Pantopaque myelogram showing characteristic filling defect at L4 on the left.
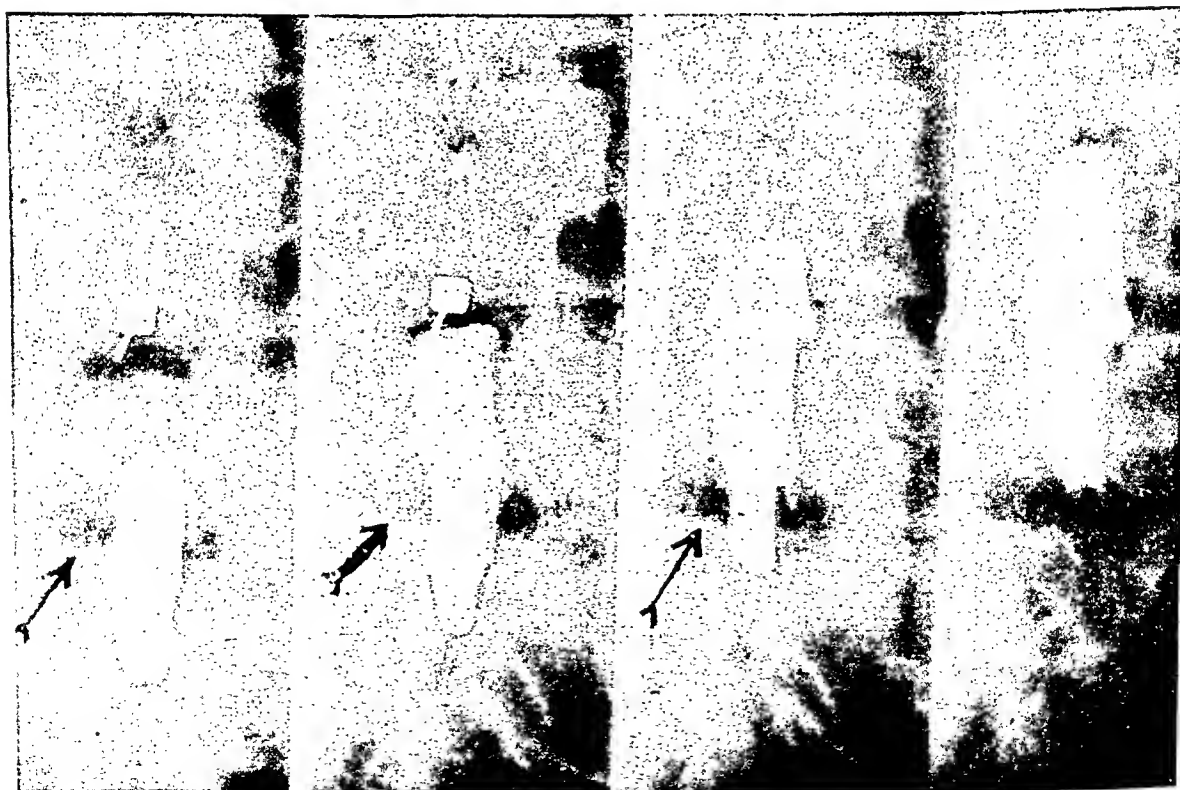
FIG. 15. Pantopaque myelogram showing characteristic filling defect at L5 on the left.

instructed in the technic of injection and in making the necessary observations while the drug is in the spinal canal, as well as in the interpretation of the spot films. Otherwise the examination may be more confusing than helpful. (Figs. 13, 14 and 15.)

It is our practice to use myelography on those patients only who are severely handicapped by low back and sciatic pain without clear-cut neurologic findings. Furthermore, the patient must have answered all of the criteria for operation. It should be stressed that myelography is not necessary for diagnosis in a ruptured disc, but it is very useful in localizing the lesion.

LABORATORY EXAMINATIONS

Many observers have recommended studying the spinal fluid in cases of suspected ruptured disc, particularly to determine if the total protein is increased. This procedure in our experience has been a totally wasted effort. Only in those cases in which the presence of a spinal cord tumor enters into the differential diagnosis do we recommend diagnostic lumbar puncture before myelography.

The sedimentation rate is an important routine laboratory study. This may be the only positive information of an early Marie-Strumpell arthritis, whereas in an uncomplicated ruptured disc the sedimentation rate is never elevated.

SURGICAL CONSIDERATIONS

Selection of Cases. Patients in their initial episode of pain, i.e., low back and sciatic pain, should have prolonged conservative treatment, for a large proportion of them will get well without surgery. The duration of conservative treatment depends on many factors, not the least of which is the economic status of the patient. A wage earner with a family to support cannot be kept flat in bed for a month or two with no assurance that he will be back to his job at the end of that time. It may be advisable in such a patient if the objective findings are well defined to cut the period of conservative treatment to two weeks, particularly if at the end of that time the patient has

shown no improvement. Ordinarily, the patients who get relief from conservative treatment show some improvement after a few days of bed rest.

Patients who have had recurrent episodes of low back and leg pain and in whom the diagnosis is unequivocal are candidates for surgery. However, it is exceedingly important never to subject one of these patients to surgery during a period of remission. They must be having severe pain at the time they enter the hospital for either surgery or myelography. Otherwise it may be difficult to localize the lesion accurately. It is our practice, no matter how typical the history, to have the patient wait for another acute bout of pain before entering the hospital for study or operation.

Low back pain alone is seldom, if ever, an indication for an operation upon a lumbar intervertebral disc. Patients with incapacitating low back disability are strictly an orthopedic problem, either for conservative treatment with braces, etc., or for spinal fusion.

Unilateral, or occasionally bilateral, sciatic pain which is refractory to conservative treatment is the only specific indication for lumbar disc surgery. Furthermore, low back and sciatic pain must be incapacitating. The mild discomfort that so many patients complain of is not an indication for either operation or myelography. Furthermore, most patients whom we consider eligible for surgery have positive neurologic signs; otherwise, they are referred to the orthopedist for conservative management.

CONSERVATIVE TREATMENT

This is an orthopedic problem and it will receive only a cursory discussion here. In our experience the most effective measures are: (1) Complete rest on a hard flat bed until the acute pain has subsided. (2) If the sciatic pain is severe, traction upon the affected leg may be beneficial. (3) A well fitting low back brace made to the patient's own measurements is helpful once the patient becomes ambulatory.

SURGICAL TREATMENT

This operation may be one of the most difficult to perform properly. It should not be attempted unless the surgeon has been thoroughly schooled in the technic of intraspinal surgery. An intimate knowledge of regional anatomy is mandatory. The operation should accomplish the removal of all the nucleus pulposus, loose pieces of posterior annulus fibrosus and cartilaginous plates. This objective is possible without destroying either the laminae or the articular facets.

It is most difficult to teach the novice to recognize the exact level at which he is operating. Almost always he will place the incision too high. To avoid this difficulty it is always advisable for the operator to identify the lowest interspace by palpating the sacrum through the open wound.

The incision should be made directly over the spinous processes centering on the level of the suspected ruptured disc. The incision should be sufficiently long to allow the operator to work in comfort. However, the experienced operator requires only a small incision. An incision 2 inches long is quite adequate. The muscles on the side of the pain are dissected from the spinous processes and a hand retractor (similar to the Hibbs retractor) is used to displace the muscles lateralward. This maneuver exposes the ligamentum flavum.

At the fourth interspace, and often at the fifth interspace, there is a ledge of lamina overhanging the ligamentum flavum. The operator should remember that the ligamentum flavum is attached to the undersurface of the lamina above and the uppersurface of the lamina below. When this overhanging ledge of lamina interferes with dissection of the ligamentum flavum, it should be removed with rongeurs up to the insertion of the ligament itself. The ligamentum flavum is then removed by sharp dissection, care being taken not to injure the spinal dura or the dura over the nerve root during the dissection. A small nasal punch is a valuable adjunct in dissecting the lateral margin of the ligamentum flavum. Once the ligament is removed the lesion should be easily demonstrated by palpation with an instrument for the imprisoned nerve root will be found pushed backward against the ligamentum flavum. The nerve root is gently dissected free from the bulging mass with pledgets of cotton and retracted gently toward the midline. The bulging mass of disc material is then removed piecemeal with a pituitary rongeur or a similar biting instrument. At this stage most operators experience difficulty with the plexus of veins on the anterior surface of the spinal canal. These are easily handled provided that certain precautions are taken during the dissection. In the first place, once the ligamentum flavum is removed the extradural fat should be gently pushed upward and downward from the nerve root, thus exposing this mass of veins. Then they should be gently pushed backward and packed away from the disc with pledgets of cotton attached to silk sutures to prevent their being lost in the wound. If a vein is accidentally ruptured during the process of packing, a piece of gelfoam saturated with thrombin may be placed on the bleeding surface and the whole mass packed upward and downward again with pledgets of cotton. This assures a perfectly dry field for the leisurely removal of the ruptured disc. Once the bulging mass has been removed the rongeurs are then placed within the disc proper and all loose fragments are excised piecemeal. During this maneuver it is exceedingly important that the operator identify the anterior longitudinal ligament or he may tear through the anterior portion of the disc and be in serious danger of damaging important structures at that level, notably the iliac arteries and veins. The dissection within the disc should be thorough but *most carefully done.* Several sharp curettes of varying shapes greatly facilitate the removal of these loose particles of nucleus pulposus and cartilaginous plates. When no further loose particles can be obtained with either the biting rongeurs or the sharp curettes, the operation is completed. We have routinely used small packs of gelfoam around the disc incision in order to mini-

mize the dangers of postoperative bleeding. We have also covered the dura mater where the ligamentum flavum has been removed with strips of gelfoam. Closure of the wound is then made in layers with sutures of black silk.

When one fails to find a frank bulging mass beneath the nerve root at the suspected level, the disc should be carefully exposed by traction on the dura toward the midline. Then the disc should be carefully palpated with a blunt instrument. Not infrequently one will demonstrate the bulging mass more toward the midline rather than in the usual lateral position; and furthermore, when such a bulging mass is palpated with a blunt instrument, it is found to be exceedingly soft and resilient. Often the blunt instrument will fall into the tear in the annulus at this site. This is positive evidence of a ruptured disc and, therefore, justifies complete radical removal of the disc.

On the other hand, one is confronted occasionally with a disc that shows no such weak places in the annulus fibrosus. The disc may feel somewhat soft to palpation but certainly one cannot demonstrate a weakened portion in it. In such circumstances we believe that the disc should *never* be opened; and if no frank rupture can be demonstrated above or below this location, the wound should be closed and the exploration listed as a negative one.

The choice of anesthesia is an individual matter. We prefer spinal procain—100 to 120 mg. This anesthetic lasts for approximately forty minutes and assures complete muscular relaxation. No supplementary inhalation or intravenous anesthetics are used.

SPINAL FUSION

The place of spinal fusion in the treatment of ruptured intervertebral discs is still a moot question. In some clinics primary spinal fusion, after the removal of a disc, is more or less a routine procedure. In other clinics it is used in only a small percentage of cases. The most usual indication for primary fusion is instability of the lower lumbar spine due to congenital anomalies. In some clinics the presence of arthritic spurs in the region of disc disorders is considered sufficient reason for a primary spinal fusion.

In our clinic we have never resorted to primary spinal fusion for ruptured intervertebral disc *under any circumstances*. We believe that combining the operation is not to the best interest of the patient because we believe that divided responsibility in a surgical case is always an undesirable factor. Our attitude is that if spinal fusion proves to be necessary in order to correct intractable low back pain it had better be done as a separate procedure. The disc operation, providing it is done skillfully, will not in any way interfere with the operative field of the orthopedic surgeon when it comes time for a spinal fusion.

In our experience relatively few patients have complained sufficiently of intractable low back pain to justify spinal fusion after disc removal. Since there is no way of predicting preoperatively which patients will have this pain, it seems undesirable to subject patients to a fusion until time has proved that simple disc removal is not enough to affect a cure. Furthermore, we have never been impressed with the fact that congenital anomalies in the lower lumbar spine play any part in disc symptomatology. We have repeatedly removed ruptured discs from the fourth interspace when there was in addition a well defined spondylolisthesis at L5. Most of these patients recovered about as normally as the patients without congenital anomalies.

The presence of arthritic spurs has never seemed to us to be an indication for spinal fusion. It would seem probable that these spurs are nature's effort to fuse the vertebrae, and once this process is completed there is likely to be good bony fusion and therefore relief of symptoms.

AFTER-CARE

The operation for ruptured intervertebral disc is quite a simple one from the patient's point of view. There is practically no disturbance of weight-bearing surfaces other

than at the site of the ruptured disc. Since a large part of the disc is removed at the operation it always has seemed to us advisable to keep the patient in bed for two weeks. Our routine is to leave the patient completely recumbent for the first twelve days, allow him to sit on the edge of the bed on the thirteenth day and to walk, on the fourteenth day at which time he returns home. He is instructed to increase his activities gradually during the next two weeks, but under no circumstances is he to put any strain upon the back such as lifting or stooping during that period. At the end of four weeks he is permitted to increase his activities and if his work is sedentary in nature he can return to his desk. Laborers and those who indulge in occupations requiring bending and lifting operations are advised to remain away from their work for three months postoperatively and then gradually to resume it. No postoperative immobilization is recommended.

Certain patients will develop excruciating back pain during the postoperative period. It will often be associated with a marked list of the pelvis and with marked muscle spasm. They are usually comfortable in bed. This type of patient in our experience usually develops in the course of time a full-blown proliferative arthritis at the diseased joint, and once the joint is fused, usually within a matter of six months, the pain disappears spontaneously.

Patients suffering in this way can often be greatly relieved by a light body cast applied with the back in extension, usually by overhead traction. This should be worn for from four to six weeks at the end of which time it may be bivalved and discarded completely in most cases. A light plaster body cast has, in our experience, been much more satisfactory than a well fitting back brace.

RECURRENT SYMPTOMS

The disc lesion recurred at the original level and was verified by reoperation in 3.3 per cent of the patients studied in this series. No doubt some of the patients listed as unsatisfactory results have recurrence of their lesion which has not yet been verified by reoperation. It seems safe to say, therefore, that the incidence of recurrence, taking into consideration other possibilities connected with this follow-up study, would not exceed 10 per cent. These recurrences have been at varying intervals from a few weeks to two or three years after the initial operation.

The results of reoperation are excellent. We have had no instance in which a ruptured disc has recurred more than once.

RESULTS

Three hundred seventy-eight patients operated upon for ruptured lumbar intervertebral discs were selected for this follow-up study. The entire group constituted "private patients" operated upon personally by one of the authors. We found it impractical to use the clinic patients for this study because of inadequate follow-up facilities. In spite of the fact that the patients were classified as "private" they represent workers from every walk of life, including fifty compensation cases.

The series is a consecutive one. No patient diagnosed and operated upon for a lumbar intervertebral disc was excluded from the study.

The investigation divided itself logically into two parts: A *long term* and a *short term* follow-up study. The long term series, from January, 1939, to April, 1942, consisted of 260 cases. The short term series, from January, 1946, to March, 1947, consisted of 118 cases. The break in continuity was occasioned by the authors' period of military service. There was no significant difference in the results of the long and short term series.*

The same surgical criteria discussed in the text of this paper were, for the most part, followed in both series of patients.

In planning the questionnaire we considered it important to ask questions so simple that no one could misunderstand

* A detailed study of follow-up statistics will be reported in a later communication.

them. They were as follows: (1) Are you able to do your usual work (the same as before your illness requiring operation)? (2) Do you consider your operation to have been successful? (3) Do you continue to have leg pain? (4) Do you continue to have low back pain?

Of the 378 questionnaires sent out, 301 had been returned at the time this manuscript went to press. Three hundred sixty-two patients apparently received the follow-up letter; sixteen of them were returned unclaimed.

Of the total series (301 patients), 242 (80.4 per cent) stated that they were able to do the same work at which they were occupied prior to operation. Forty-five (14.9 per cent) stated that they were able to do their usual work part of the time but were handicapped by recurrent symptoms at other times. Fourteen patients (4.7 per cent) had to change their occupations because of persistent symptoms.

Of the total group (301 patients), 243 (80.8 per cent) considered their operation to have been successful. Twenty-nine (9.6 per cent) of them considered their operation partially successful, and the remainder, 29 (9.6 per cent) considered it unsuccessful. It is interesting to note some of the reasons given for considering the operation partially successful. One patient writes: "I can't take long automobile trips because my back gets tired." But for the most part patients who considered the operation partially successful were those who had definite episodes of recurrent low back or leg pain which temporarily incapacitated them.

One hundred forty-two patients (47.2 per cent) stated that they have no leg pain whatsoever. One hundred thirty-nine patients (46.2 per cent) continue to have an occasional mild pain in the leg but not severe enough to be incapacitating. Twenty patients (6.6 per cent) stated that they have aggravating leg pain constantly, in fact just as bad as before they were operated upon.

It is interesting to note that the question with respect to low back pain brought only 117 replies (38.9 per cent) claiming complete and lasting relief of this symptom. One hundred sixty-three patients (54.2 per cent) stated that they have occasional recurrences of low back disability although in most instances it is not incapacitating and is of short duration. Twenty-one patients (6.9 per cent) claim to be completely incapacitated for their normal activity by persistent low back disability.

We consider the two questions, (1) Are you able to do your usual work, and (2) Do you consider your operation to have been successful, to be of paramount importance in attempting to evaluate the results of lumbar disc surgery. As we have pointed out elsewhere in this paper no surgical effort can hope to restore a ruptured disc to a normal functional state. It is not surprising, therefore, that somewhat less than half of the patients have been completely free of all symptoms referable to their former disc disturbance since operation. That over 80 per cent of the entire group of patients are able to carry on their usual activities and are satisfied with their operation (for which in most instances they paid out of their own savings) speaks eloquently for the practical aspects of conservative disc surgery.

OPERATIVE MORTALITY

One patient of this series of 378 cases died on the eighth postoperative day; an operative mortality of .26 per cent. Death was due to fulminating influenzal pneumonia (confirmed by necropsy). Since the death occurred in February, 1939, the newer sulfa drugs and penicillin were not available for treatment.

It would seem logical to assume, therefore, that the operative risk for lumbar intervertebral disc surgery is no greater than that for non-complicated inguinal hernia.

SUMMARY

1. A thorough knowledge of the anatomic relations in the lower lumbar portion

of the spine and of the physiology of the intervertebral disc is essential for a clear understanding of the clinical features of this disorder. The pathologic changes which occur in a ruptured lumbar intervertebral disc are irreversible; no surgical effort can hope to restore its anatomic integrity.

2. We believe that the term "ruptured intervertebral disc with or without herniation of the nucleus pulposus" more nearly describes the anatomic and pathologic features of the disorder than any other. The term "hidden disc" seems to be an unfortunate one and in our experience usually represents a misnomer for a negative exploration. Multiple ruptured discs are rare in our experience (2 per cent).

3. Low back pain, uncomplicated by sciatic radiation, is seldom if ever an indication for operation upon a lumbar intervertebral disc.

4. Unilateral (occasionally bilateral) sciatica which is refractory to conservative treatment is the only specific indication for lumbar disc surgery.

5. Patients in their initial episode of pain should have prolonged conservative treatment. A large proportion of them will get well without surgery.

6. Most patients with ruptured lumbar intervertebral discs whose symptoms are severe enough to justify operation present unequivocal neurologic signs. Not only are these signs invaluable and accurate in diagnosis but they can be relied upon in over half of the cases for localization of the lesion.

7. No matter how classical the history of a ruptured intervertebral disc may be, a patient should never be operated upon in a remission. This is one of the most common causes for negative explorations.

8. Disc operations may be among the most difficult in surgery to perform properly. The operation should accomplish removal of all the nucleus pulposus, loose pieces of annulus fibrosus and cartilaginous plates.

9. Myelography is not required for ... should be made on clinical

findings alone. However, myelography is extremely valuable in localizing the lesion when the neurologic signs are not clear-cut.

10. Primary spinal fusion together with simple removal of the ruptured intervertebral disc is not a desirable procedure. Those patients who continue to have incapacitating backache after simple disc surgery may require a secondary spinal fusion. A disc operation properly performed does not in any way interfere with subsequent fusion operations.

11. The follow-up results of 301 verified ruptured intervertebral discs treated surgically are presented.

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MÉNIÈRE'S DISEASE*

ITS SURGICAL TREATMENT BY DIVISION OF THE ACOUSTIC NERVE

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MÉNIÈRE'S disease, or Ménière's syndrome, is characterized by paroxysmal attacks of vertigo in the presence of tinnitus and impaired hearing which are usually unilateral. Ménière's classical description of the syndrome which bears his name was made in 1861;[1] and although the disease is fairly common, the etiology is still undetermined and ideas of treatment vary widely. The symptoms are of varying degree and the milder ones are either tolerable or possibly benefited by medical therapy. But the more severe and intractable form of the disease is best treated by division of the acoustic nerve.

This report deals with fifty consecutive cases in which the operation has been employed and the patients. followed thereafter from one to eight years.

PATHOLOGY AND ETIOLOGY

Since Ménière's disease is of itself not fatal, there is a paucity of autopsy material for the study of the pathogenesis. Hallpike and Cairns[2] in 1938 made the first report on the histologic changes in the temporal bones of two patients who had had typical Ménière's disease. In each of these cases they described gross distention of the endolymphatic system together with degenerative changes in the sensory elements. Since then, there have appeared other reports[3-7] with comparable postmortem findings of alteration in the endolymphatic system. It is inferred from these reports that the dilatation of the endolymphatic system results from overproduction of endolymph, from its defective absorption, or a combination of both, and that the location of the lesion in Ménière's disease is in the inner ear. Others, particularly Dandy,[8] have maintained that the lesion is not located here but along the afferent pathway, probably in the acoustic nerve.

The sensory end organs in the inner ear are in the semicircular canals, the maculae of the utriculus and the sacculus, and in the organ of Corti of the cochlea. The vestibular portion of the acoustic nerve coming from the semicircular canals and the maculae, and the cochlear portion from the organ of Corti come together near the inner end of the internal auditory canal and for a few millimeters have a common sheath. Near the brain stem the trunk of the nerve divides again into vestibular and cochlear portions which pass separately to their respective nuclei in the medulla. Nowhere do the central pathways of these two nerves come in close proximity.

Crowe[9] concluded that a single lesion in the brain stem or cerebrum could not cause all the symptoms of Ménière's disease without involving other structures and that anatomically there are only two possible sites in which a single lesion could cause the symptoms: in the nerve trunk or in the inner ear. Of the two he favored the latter and pointed out that although Corti's organ and the vestibular end organs are widely separated, they have the endolymphatic system in common. Dandy,[10] on the other hand, who shared with Crowe the responsibility for the care of many patients with Ménière's disease, maintained that the lesion lies in the acoustic nerve and not in the end organs and that a lesion affecting both cochlear and semicircular canals would be highly unlikely. He considered Ménière's disease similar to trigeminal neuralgia, also a paroxysmal disease and believed to result from a lesion in or on

the sensory root of the trigeminal nerve. Horrax[11] and Colemen and Lyerly[12] have concurred in this idea.

Although no gross or impressive microscopic alteration of the acoustic nerve has been found in autopsy specimens or in segments of the nerve resected at operation, the suggestion has been made[10,13] that pressure on the nerve or intrinsic changes in it may result from anomalous vessels, chiefly the anterior inferior cerebellar and auditory arteries. These arteries show great variability in their size, configuration and relation to the nerve but their rôle in the etiology of the disease is difficult to establish. That vertigo and tinnitus may arise from the acoustic nerve can be demonstrated by low voltage faradic stimulation of the exposed nerve in a patient awake. Also, that all three symptoms of the Ménière's syndrome can occur from invasion or compression of the nerve is demonstrated by such lesions as the acoustic nerve tumor and the aneurysm of the vertebral artery. Nevertheless, it is also true that such lesions are not found to produce the typical paroxysms of vertigo so characteristic of Ménière's syndrome.

Infectious or "toxic" neuritis of the acoustic nerve which has occasionally been suggested as possibly responsible for Ménière's syndrome seems unlikely in view of the chronic course of the symptoms and the absence of changes in the nerve. In addition, there is a notable lack of infectious or "toxic states" in patients with Ménière's disease.

A frequent argument given to refute the contention that the disease has its origin in the inner ear is that tinnitus may persist after total division of the nerve but I have also observed persistence of tinnitus after total removal of a tumor of the acoustic nerve and division of the nerve proximally. A plausible suggestion made by Atkinson[14] is that tinnitus may be considered a form of paresthesia which may persist even after the nerve is cut and regardless of where the original lesion lies. Others contend that since destruction of the inner ear by surgical means will abolish symptoms, or at least vertigo, this precludes the possibility of the lesion's existence in the nerve. But in dispute of this it may be recalled that in trigeminal neuralgia, a condition in which no one believes the lesion exists in the sensory end organs, the pain can be relieved by procainization of the superficial area in which the pain is felt. Thus none of the rationale for locating the lesion in Ménière's disease in either the end organs or the afferent pathway is entirely valid. Nor is reasoning clarified by the occasional encounter of a patient who has paroxysmal vertigo and tinnitus from an ear that is totally deaf and lacks evidence of response to vestibular tests, yet is relieved by intracranial division of the auditory nerve.

Empirical reasoning based on the beneficial results claimed for various forms of medical treatment has colored much of the discussion concerning the etiology of Ménière's disease. Atkinson,[15] in dealing with this aspect of the problem, has suggested that a basic fault, which can be influenced by a variety of medical treatments, could be an alteration in capillary permeability particularly in capillaries of the stria vascularis in the labyrinth. This explanation previously advanced by Brunner[16] and Portmann[17] is thought to have received support from the autopsy findings of dilatation of the endolymphatic spaces. Atkinson[15] suggests further that an increase in capillary permeability can be produced either by anoxia from a diminished blood supply which impairs the function of the capillary walls or by sensitization to some foreign protein or chemical. In either case the resulting alteration of the fluid content of the endolymphatic spaces might be the source of symptoms and of the reported changes in these spaces. While it seems to follow that periodic alterations in capillary permeability might coincide with paroxysms of vertigo and fluctuations in tinnitus, some organic changes must also take place to cause the loss of hearing and sometimes loss of response to vestibular tests.

In the fifty cases reviewed for this report, the disease occurred in thirty-three males and seventeen females, a ratio of two to one. The disease was exclusively or predominantly on the right side in twenty-six patients and on the left in twenty-four. It is largely a disease of middle life as is shown by the age of onset of first symptoms. In the first two decades there were no patients; in the third, eleven; in the fourth, sixteen; in the fifth, fourteen; in the sixth, six; and in the seventh, three. The youngest age at which symptoms began was twenty and the oldest sixty-two. These ages do not correspond to the ages which patients attained before coming to operation and while eleven developed their first symptoms in the third decade only three came to operation before the age of thirty, and there were five after the age of sixty.

Scrutiny of the patients' past histories failed to show with the possible exception of a few cases that the onset or progress of the disease might be significantly related to other diseases or to injuries. One dated the onset of unilateral deafness and tinnitus later followed by vertigo to a deafening explosion and a simultaneous head injury and concussion. Another dated the onset of vertigo to severe epidemic influenza (1918) and had comparatively frequent attacks of vertigo for the next twenty-two years. Still another experienced the simultaneous onset of vertigo, unilateral tinnitus and loss of hearing while recovering from an unidentified meningitis. Four had middle ear infections during childhood many years before the onset of Ménière's syndrome; one had a mastoidectomy on the side of Ménière's disease fifteen years before, and one elderly patient had a greatly sclerosed mastoid bone on the side of the disease. The general physical examinations and routine laboratory studies of all patients showed two with mild diabetes mellitus, three with mild hypertension, one with rheumatic heart disease and six with recurring paranasal sinusitis seemingly unrelated to Ménière's disease. There was a conspicuous absence of other diseases of the nervous system, tuberculosis, syphilis, blood dyscrasias, metabolic diseases, vasospastic and vascular occlusive diseases and of allergic disorders such as hives, asthma and intolerance of specific foods and drugs. There was, however, a comparatively high incidence of headache, some typically migrainous but mostly of a special variety which might be considered as a symptom directly or indirectly related to Méinère's syndrome.

SYMPTOMATOLOGY

The cardinal symptoms of Ménière's disease are paroxysms of vertigo, impaired hearing of the inner ear type which is usually unilateral and grows steadily worse, and tinnitus which is referred to the deaf ear. Aside from the tinnitus and deafness the patients usually feel perfectly well between the attacks of vertigo. In some patients all three symptoms have a simultaneous onset which makes early recognition of the disease relatively simple but this occurred in only 18 per cent of my cases. In other cases any one or a combination of two of the symptoms antedated the other symptom or symptoms by weeks, months or years. In 28 per cent of the cases unilateral tinnitus alone antedated the other symptoms, the longest interval being fifteen years. In 24 per cent vertigo alone antedated the other symptoms, the longest interval being ten years. In 16 per cent unilateral impairment of hearing alone antedated the other symptoms, the longest interval being twelve years before tinnitus developed and, in the same patient, eighteen years before vertigo occurred. In 14 per cent various combinations of two simultaneously occurring symptoms antedated the appearance of the third symptom, the longest interval being six years.

From these data it is evident that incipient Ménière's disease often exists before the advent of the characteristic triad which makes recognition of the disease possible or reliable.

In eight cases or 16 per cent of this series there was bilateral tinnitus in addition to vertigo and hearing loss; and while it must

be recognized that bilateral Ménière's disease occurs in about this proportion, a special problem exists in these cases if unilateral nerve section is contemplated. Usually, there is some indication that one side is more involved by the disease than the other and this, from my experience, is the clue to the location of the offending side as far as the attacks of vertigo are concerned. In one case of bilateral tinnitus of equal degree the deafness was unilateral. In four cases of bilateral tinnitus the hearing loss was bilateral but greater on one side. In two cases of bilateral deafness of equal degree and bilateral tinnitus, the tinnitus was distinctly greater on one side. But in one case in which the bilateral tinnitus and bilateral deafness were each of equal degree the reliable clue to the location of the offending side, as far as vertigo is concerned, appears to have been a "dead" labyrinth on that side. These conclusions seem justifiable in view of the successful outcome of unilateral acoustic nerve section in each case. There were no cases in which bilateral deafness and unilateral tinnitus occurred.

Vertigo. Vertigo is the most striking and most distressing feature of Ménière's syndrome and the symptom for which patients chiefly seek relief. The sensation is one in which surrounding objects seem to whirl or jump in any direction within a horizontal or vertical plane. However, the direction and plane are of no value in determining from which side the trouble arises. As Crowe[9] has recalled, the discrepancy in the direction of the vertigo can be reproduced in normal persons by alternating the temperature of the water in the caloric test or alternating the direction of the rotation in the Barany tests. The vertigo characteristically recurs in attacks that are usually sudden, sometimes so sudden that the patient is thrown to the floor or is rapidly propelled off balance. Three of the patients of this series had sustained fractured bones in falls during attacks. The attacks last from a few seconds to many hours and occasionally for several days;

the longest attack in a patient's experience is likely to be his first one. During an attack the patient must support himself or lie down; and though some find benefit from lying motionless on one side or the other, unrelated to the side of the disease, the attack for the most part seems to run its course unaffected by position. Two patients reported that they could suspend the vertigo as long as they could forcibly deviate the eyes in a special direction. In one of these observed during an attack, deviation of the eyes away from the direction of the nystagmus was said to suspend the vertigo but the patient was unable to hold the eyes in deviation for more than a minute at a time.

The intervals between attacks of vertigo vary in different patients and in the same patient. It is the unpredictability as well as increasing frequency of the attacks which bring most patients to consider operation. Spontaneous recovery from attacks undoubtedly does occur sometimes and similar to the attitude that patients with trigeminal neuralgia often take, there is the hope that each attack may be the last. In this series eight patients had remissions from vertigo for a year or more and one for fifteen years, but after these long remissions the frequency of the attacks steadily increased. In the early part of the disease the patients are usually free of any sense of vertigo or of sensations of imbalance between attacks though some in the later period of the disease have a more or less constant feeling of giddiness, unsteadiness or swaying which may be accentuated by movements of the head or sudden changes in position of the body. But patients who complain only of dizziness, swooning, giddiness, unsteadiness or uncertain sensations in the head without definite episodes of vertigo do not have Ménière's disease and this feature alone can be relied upon to differentiate these commoner complaints from those of Ménière's disease.

While in the majority of cases there are no aura preceding attacks of vertigo, in six patients (12 per cent) there were premoni-

tory symptoms present from a few minutes to several hours which included increased tinnitus, increased impairment in hearing, a feeling of fullness or aching in the head and ear on the side of the lesion, flushed feeling in the face on the side of the lesion, numbness in the hands and throat and faintness.

In all but two patients vomiting occurred with vertigo though in the majority it accompanied only the more severe attacks of vertigo. It was not preceded by nausea in some and a few denied ever experiencing nausea even in prolonged attacks of vertigo with vomiting. Some patients were so impressed by the vomiting that they tended to minimize the vertigo and attributed the entire episode to "upset stomach." Other symptoms which are commonly recognized as accompanying nausea and vomiting from any cause and need not be considered as peculiar to a Ménière's attack included, chilliness, sweating, headache, palpitation, borborygmus and diarrhea.

Tinnitus. Tinnitus of Ménière's disease is not any different in its characteristics from that which occurs from many other conditions, whether in the peripheral mechanism or central pathway for hearing. It was present in all cases of this series and bilateral in eight. The term "tinnitus" strictly speaking means a ringing, but in common usage it includes sounds variously described as hissing, rushing, buzzing, sizzling, whistling, swishing, screaming, roaring, beating, drumming, vibrating or explosive. There is great variation in quality, pitch, intensity and in frequency of alternating sound even in the same patient and the purely ringing sound may be described as humming, singing, tinkling or gong-like. A sailor was particularly distressed because his tinnitus was often indistinguishable from a bell buoy and a typist could not always distinguish hers from the warning bell on her typewriter.

There are two modes of onset of tinnitus in Ménière's disease. One is abrupt and loud and usually persistent, and is frequently attended from the start by impaired hearing. The other is gradual and more or less intermittent but steadily increasing in pitch and frequency with the passage of time. In about half the patients studied there was some increase in the tinnitus noticed before, during or after attacks of vertigo.

While tinnitus constitutes one of the complaints in patients with Ménière's disease, it is not the predominant complaint. In my experience most patients confronted with the decision of an operation intended to relieve vertigo but not promised to relieve their tinnitus show comparatively little concern over the prospect of continuation of their tinnitus if vertigo is relieved. In patients with or without Ménière's syndrome who are totally deaf or in a state of anxiety tinnitus may become an obsession.

Deafness. Loss of hearing in Ménière's disease has all the characteristics of deafness of a cochlear or acoustic nerve lesion. Both air and bone conduction are impaired and although the audiometric curves are variable, in most instances all tones are more or less affected. The hearing loss is not usually total but in most patients that come to operation the hearing remaining on the side from which attacks arise is of little practical value. Like tinnitus, the deafness may have an abrupt or a gradual onset with an insidious progression that makes it difficult for the patient to be certain of its beginning and sometimes unaware of its presence. After it progresses to a point beyond useful hearing and short of total deafness it usually remains stationary. Temporary increase in deafness is often evident at the time of an attack of vertigo. Only three patients had relatively normal hearing by audiometric tests and none of these had a complaint of deafness. In the majority, the loss was between 35 and 60 per cent in the diseased ear which is in a range below useful hearing. Seven patients had significant loss of hearing in both ears.

Other Symptoms and Signs. The occurrence of some form of headache in many of the patients with Ménière's disease has led a few to suggest that the two condi-

tions may have a common etiologic vascular mechanism. In this series, six of the patients had been subject to recurrent headaches prior to the onset of the first symptom of Ménière's disease and this is about the percentage in which headaches occur in the general populace. The incidence of headache after the advent of Ménière's syndrome was considerably higher (about 50 per cent) but includes a variety of headaches. In many it was bilateral or generalized and not impressively related in time of occurrence to the attacks of vertigo and considered to be probably a manifestation of an anxiety state based on a justifiable fear of unpredictable attacks of vertigo. There were other patients (18 per cent), however, with symptoms about the head more suggestive of some direct relationship with Ménière's disease. These symptoms included a sense of fullness, pressure or aching and even sharp pain in the ear, about the ear or some part of the head on the side of the disease which preceded, accompanied or followed an attack and sometimes occurred between attacks. A few mentioned a sense of flushing and even aching in one side of the face prior to an attack. Three described a feeling of a "dead cheek" occurring on the side of the tinnitus and deafness, particularly with attacks of vertigo and two of these appeared to have demonstrable sensory impairment in the side of the face including diminished corneal sensation. In these latter cases the numbness disappeared after division of the acoustic nerve.

Visual disturbances including blurring, diplopia and hallucinations of flashes, rings or scotomas occurring during and after an attack of vertigo were reported by nearly half the patients but with the exception of the hallucinations the symptoms might readily be explained on the basis of the nystagmus which accompanies the attacks.

Loss of consciousness occurring for short periods with the onset of more severe attacks of vertigo was reported by four patients. In each of these there has been no recurrence of loss of consciousness following relief of the attacks of vertigo by operation, which suggests that the syncope may have resulted from fall in blood pressure that came with the attacks of vertigo.

DIAGNOSIS

The diagnosis of Ménière's disease is made almost wholly on the history and should not be difficult if attention is given to the description given by the patient and if the triad of symptoms is present, namely, attacks of vertigo, tinnitus and deafness. While vertigo alone may be a manifestation of the disease, from a surgical standpoint the diagnosis cannot be made until tinnitus or deafness or both develop. In this series every patient had all three symptoms. Nystagmus undoubtedly occurs with every attack of vertigo (although I have observed few attacks) and in some it persists for a while after an attack but even when its direction is observed no definite clue can be gained as to the side of the lesion. McKenzie[18] quotes the case of a physician who observed nystagmus on himself on frequent occasions during attacks and found variation in different attacks.

The vestibular tests are not always reliable as an index of function for the results seem to vary in some patients, but the caloric test was performed one or more times in thirty-eight patients of this series. The method suggested by Atkinson[19] was used routinely but in those in whom this test produced no response more prolonged irrigation of the ear was employed. The results showed that of the patients tested the responses were normal in 35 per cent and absent on one side in 12 per cent; by comparison of the two sides there was a diminution of vestibular response in 33 per cent and an increase in 20 per cent on the side of the lesion. These figures show that the alteration in caloric response is of little value in the diagnosis yet there was one case in the series in which the test seemed to prove useful. This was the case already referred to in which tinnitus and

deafness were equal bilaterally and the absence of caloric response gave the clue to the side on which successful operation was performed.

In differential diagnosis there are few other diseases to be confused with the complete syndrome of Ménière's disease. The only diseases of importance are the cerebello-pontile angle tumors and usually there are other signs that make the diagnosis unmistakable. Dandy[10] reported encountering three unsuspected tumors when operating for Ménière's disease but no difficulties should arise from such a mistake since the operative exposure is the same for both lesions.

MEDICAL TREATMENT

A variety of medical treatments for Ménière's disease has been employed over the years, and anyone attempting to evaluate them quickly finds himself at a loss. The subject has been reviewed several times in recent years.[20,21,22] All of the current medical treatments are claimed to be predicated on clinical experiments made by their originators and the usual criteria taken for success is relief of vertigo though improvement in tinnitus and occasionally in hearing is also reported. Good results are reported to occur in from 62 to 96 per cent of patients;[22] and while the figures seem suspiciously high, undoubtedly some of the claims for improvement must be justified. Whatever the figures are it is only fair to conclude in our present state of knowledge of the disease that most patients should be given a trial period of treatment before resorting to surgery.

One method of treatment restricts fluid intake (Mygind and Dedering)[23] and another (Furstenberg)[24] attempts to accomplish the same end by dehydration with large doses of ammonium chloride; others seek to alter the electrolytic balance (Talbott and Brown)[25] while still others attempt to produce vasodilation with histamine (Horton and his collaborators)[26,27] and nicotinic acid (Atkinson)[28] or to produce desensitization by histamine

(Atkinson).[29] Perhaps each method has some advantage in particular cases.

In this series 70 per cent of the patients had some form of medical treatment and over half of them had intensive treatment before coming to operation.

SELECTION OF CASES FOR SURGERY

The indication for operation in patients with Ménière's disease is based on the frequency and severity of the attacks of vertigo and the degree of disability produced. The majority of patients with Ménière's disease do not require surgical treatment since (1) the symptoms are mild or infrequent; (2) sedatives or some more specific form of medical treatment may be useful; (3) the attacks may in time subside spontaneously or (4) some co-existing disease may make the risk of an operation unjustifiable.

No patient should be considered for operation unless he has had the disease long enough to make it possible to rule out labyrinthitis or any other condition which may be confused with Ménière's disease in the early stages. In this series the shortest interval between the onset of vertigo and operation was ten months, the longest twenty years, and the average interval was four and a half years.

In addition to vertigo a lateralizing symptom or sign, namely, unilateral tinnitus or deafness must exist. In case of bilaterality of the tinnitus or deafness there must be a predominance on one side to indicate the side on which the operation is to be performed. In case the operation is to be performed on the only side in which useful hearing remains, consideration must be given to the possibility of additional loss of hearing in the better ear from the operation.

But in the last analysis decision for operation should be entertained only when the patient's comfort or ability to carry on his occupation is compromised by his attacks of vertigo. Relief of tinnitus is not a predictable outcome of the operation and hearing is not improved. Whereas the

operation of division of the auditory nerve intracranially should carry a low mortality (less than 1 per cent), it is none the less a major operation and carries a potential risk to life.

SURGICAL TREATMENT

Historically it is interesting to note that in 1874 Charcot[30] in demonstrating cases of Ménière's disease observed that when deafness became complete the attacks stopped spontaneously and he was prompted to suggest that division of the acoustic nerve might be of benefit. Ballance,[31] in 1894, also suggested division of the eighth cranial nerve for the relief of vertigo. Krause was credited by Frazier[32] with having sectioned the eighth nerve in 1905 for the relief of tinnitus. In 1908 both Frazier[32] and Ballance[33] sectioned the nerve, one for the relief of dizziness and the other for "painful tinnitus." Frazier[34] again performed the operation in 1913 for tinnitus. None of these early operations was attended by striking success, apparently because the cases were not ideally suited and the results did not make enough impression to bring the operation into general use.

The operation of intracranial division of the acoustic nerve for Ménière's disease has been popularized by Dandy[8] who made his first report of successful results following total section of the nerve in 1928. There have followed in the succeeding years equally enthusiastic reports on the operation by Coleman and Lyerly,[12] Cairns and Brain,[35] Olivecrona,[36] Munro[37] and Adson.[20] McKenze,[38] in 1931, began partial section of the nerve designed to interrupt the vestibular fibers but spare the cochlear fibers, and Dandy[39] reported on a similar procedure in 1930, in which he suggested section of five-eighths of the cephalad portion of the acoustic nerve. Dandy,[40] in 1935, reported the successful treatment of bilateral Ménière's syndrome and recommended division of the vestibular portion of each nerve. Although no other neurosurgical clinic has had occasion to perform division of the acoustic nerve for Ménière's disease in numbers even remotely approaching those reported by Dandy (401 cases in 1941[10] and over 600 cases in 1945[41]), the operation is widely accepted by neurosurgeons here and abroad as being well suited to selected cases.

Other less commonly performed surgical forms of treatment of Ménière's disease have been directed toward the end organs of the inner ear. Portmann's[42] operation on the endolymphatic sac has relieved some patients (Waltner)[43] but the relief has often been only temporary. In England, a commonly used operation has been alcohol injection into the labyrinth (Wright)[44] either through the drum and oval window or through a trephine opening in the horizontal canal. This procedure causes destruction of both the cochlea and vestibule and sometimes causes facial paralysis. Putnam[45] reported two cases in which he coagulated the vestibule through an opening into the superior semicircular canal by way of a transtemporal approach but the approach seems unnecessarily difficult. Ablation of the membranous organ of the horizontal canal or vestibule by mechanical destruction or alcohol injected through the mastoid route has been reported by Mollison[46] and Cawthorne.[47] These procedures also destroy both cochlear and vestibular function. The procedure reported by Day[48] employs the same mastoid approach but seeks to destroy by coagulation only the vestibule and preserve hearing. By comparison with these operations on the inner ear, it appears that intracranial total or subtotal division of the acoustic nerve is more direct and selective and, as a result, more reliable in its results.

Technic of Intracranial Division of the Acoustic Nerve. In all but three cases of this series the anesthesia employed was ether vapor administered through an endotracheal tube; in the others local procain infiltration of the scalp was used. The horizontal cerebellar position was used routinely although the upright position recommended by Adson[20] may have advantages. The in-

cision in the scalp was unilateral and semi-circular with downward reflection of a soft tissue flap sufficient to give a good exposure of the occipital bone on the side of the lesion. Others recommend a straight vertical lateral incision for it can be made with slightly more speed and the occipital nerve may be preserved, but in case difficulties arise the exposure permitted by this incision may be inadequate. A window is made in the occipital bone which borders on the mastoid cells and is of sufficient size to expose the lateral half of the lateral venous sinus. If mastoid cells are accidentally or deliberately opened, they may be sealed with bone wax and additional safety provided by administering penicillin postoperatively. The dura is opened and fluid released from the cisterna magna through a small puncture in the arachnoid. This permits easier retraction of the cerebellum in exposing the region of the cerebello-pontile angle. The release of additional fluid from the lateral cistern in the angle facilitates additional retraction of the cerebellum for exposure of the acoustic nerve. The retractor should be as close and as nearly parallel as possible to the tentorium. Any veins which pass from the cerebellum to the tentorium that may be torn by the retraction should be coagulated. Small vessels, either arteries or veins, about the nerve should be gently brushed aside or coagulated and divided if necessary in order to obtain clear exposure of the nerve. Occasionally, the facial nerve can be partly seen beneath the cephalad border of the acoustic nerve but usually it is desirable to separate the two gently with a nerve hook. Separation of the two nerves is not always easy but is necessary in order to avoid damage to the facial nerve and facial paralysis. While the hook lifts the acoustic nerve, the nerve can be partly or wholly divided with a knife or the hook may be touched with the cutting current of the electrosurgical unit. In partial division of the nerve when it is desired to interrupt only the vestibular fibers and preserve the major portion of

the cochlear fibers, the anterior or cephalad half, or slightly more, of the nerve is cut. In the closure of the wound it is desirable to approximate the dura accurately and minimize leakage of cerebrospinal fluid by sealing off the line of closure with gelatin foam strips. The soft tissue flap is restored to its position and accurately approximated in layers with interrupted silk sutures. Only a covering dressing for the wound is necessary though I have found some advantage in maintaining moderate pressure over the area for a day or so by sponge rubber held in position with a gauze roll encircling the head. This serves to minimize the collection of fluid and blood between the dura and muscle. When properly done, the operation carries very little risk and the patient can usually get out of bed on the second or third day.

Results of Operation. Unilateral operations were performed in all patients of this series even in those who had bilateral symptoms of one kind or another and thus far there has been no need for considering division also of the nerve on the opposite side. One patient of sixty-two years died from a cerebral hemorrhage which occurred on the fifth postoperative day and was verified at autopsy. One patient developed a complete facial paralysis which, however, recovered after several months; it resulted apparently from the manipulation necessitated in the control of bleeding from a torn petrosal vein. Three patients developed very slight facial weakness which disappeared within two weeks after operation.

In twenty patients partial division of the nerve was performed and in thirty the entire nerve was divided. All but two were completely relieved of vertigo. The two that were unrelieved had partial nerve sections and by postoperative caloric tests were found to have vestibular fibers remaining. In one of these a total division of the nerve was performed three months later with complete relief of vertigo. The other patient found that her attacks of vertigo, though persistent now for seven

years postoperatively, have been significantly diminished in frequency and degree and she has been unwilling to submit to a second operation. As a result of this experience it is my conclusion that failure of the operation to relieve vertigo in properly chosen cases of Ménière's disease (at least in unilateral disease) results from incomplete division of the vestibular fibers. For this reason total division of the nerve is preferable unless there is some special reason for preserving the residual hearing in the diseased ear.

In the early postoperative period there is usually a complaint of more or less dizziness which I have rarely found to be described as vertigo. Associated with this there is usually nystagmus of moderate degree when the eyes are deviated away from the side of the operation. Blurred vision difficulty in focusing and occasionally diplopia are also frequent complaints in the early stages after operation. But all of these subside steadily and are usually gone or are minimal within a few days to several weeks. Only a few patients will mention these disturbances for longer periods of time and none in this series was incapacitated by them after the initial period of recovery from operation. In all, the Rhomberg test was negative. Patients who have a dead labyrinth prior to operation are likely to be troubled very little or not at all by these symptoms. The majority of patients resumed their normal activities in from three weeks to three months after operation.

Tinnitus is unfortunately not relieved in all patients but may be improved and is rarely worse. In those in whom the tinnitus is improved but not eliminated there is less intensity and lowering of the pitch. Greater improvement in tinnitus followed total division of the nerve than followed the partial division. In the patients having total nerve division tinnitus was abolished in 30 per cent, definitely improved in 40 per cent and unimproved in 30 per cent. In patients having partial nerve division tinnitus was abolished in only 15 per cent,

improved in 25 per cent and unimproved in 60 per cent. This discrepancy in the effect of the two types of operation on tinnitus is perhaps an additional reason for favoring total division of the nerve.

Hearing in those patients subjected to partial division of the nerve was not found after operation to be improved by audiometric tests though some believed they could hear better particularly if tinnitus was diminished or eliminated. But some of the patients with total unilateral nerve section also expressed the belief that their ability to hear was improved and only a few believed their useful hearing to be worse. It is a common belief that residual hearing following partial section of the nerve is progressively lost thereafter but this has been actually observed by symptom and audiometric tests in only one patient of this series.

SUMMARY AND CONCLUSIONS

Patients with Ménière's disease may be benefited by medical treatment; but if the treatment is ineffective, they should be advised to choose operation. Other patients whose occupation may make the risk of an attack of vertigo hazardous or whose economic status does not permit the loss of time should be similarly advised.

Of the several forms of surgical treatment intracranial division of the acoustic nerve gives the best results.

A review of fifty cases in which total or partial intracranial division of the acoustic nerve was performed shows that in a follow-up period of one to eight years all but one was relieved of vertigo.

Failure to relieve vertigo in unilateral Ménière's disease is due to incomplete division of the vestibular fibers.

Except when it is especially desirable to preserve hearing total division of the acoustic nerve has an advantage over partial division in that it is easier to perform, all vestibular fibers are unquestionably cut and there is a greater likelihood of obtaining relief from tinnitus as well as vertigo. Usually patients with hearing

already impaired by Ménière's disease do not find total deafness in the diseased ear an increased handicap.

The morbidity and mortality of the operation are not formidable.

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PONTINE tumors produce extensive cranial-nerve disturbance, usually early and bilateral. Pyramidal-tract involvement is greater than in other posterior fossa tumors.

ABSCESS OF THE BRAIN*

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THE incidence of brain abscess formation in recent years has decreased tremendously. The disease, however, is still common enough to command a continued interest in this subject. The literature over the past fifty years is voluminous and a review of these articles leaves little or nothing new to be added. A few of these outstanding publications may be classed as surgical milestones in the treatment of brain abscesses and as such justify calling attention to them again.

It is believed that the same therapeutic agents that have been responsible for the total reduction in the number of brain abscesses have also served to alter some features of the symptomatology as previously recognized in these lesions. With a lull in the acute symptoms, a false security, thwarted with serious potentialities, has resulted. In the past five years, newer surgical ventures fortified with newer therapeutic agents offer some advantages and more assurance of a cure over previous methods of treatment. The experiences set forth in this communication are based on a study of fifty-six patients with abscesses located within the cerebral or cerebellar tissues. An attempt to depict the symptomatology and evaluation of the various types of treatments instituted in these fifty-six personal cases will be undertaken after a brief reference to the more outstanding surgical contributions on this subject. (Table 1.)

It would be short of sacrilegious to develop a monograph on the subject of brain abscesses without tribute to the work of Macewen.[1] In his selected cases published in 1893, he charts ninety-four patients with infective intracranial lesions in which seventy-four were operated upon; sixty-three were "cured" and thirty-one died. Of twenty-five cases of intracranial abscess, nineteen were operated upon and eighteen were "cured." One death occurred. This contribution set the score which neurosurgeons have since attempted to equal. Dow-

TABLE 1
FIFTY-SIX PATIENTS WITH ABSCESS OF THE BRAIN—
FOURTY-SEVEN SURGICALLY TREATED WITH
EIGHTEEN DEATHS

	No. of Cases	Deaths	Living
Autopsy (No Surgery)	9	9	0
Aspiration (Only)	12	5	7
Aspiration (Later Drainage)	2	2	0
Drainage (Only)	19	6	13
Decompression and Aspiration	3	2	1
Resection	9	1	8
Decompression (Only)	2	2	0

man[2] advocated in 1923 the production of adhesions between the cortex and the dura before the drainage of an underlying abscess. In 1924 King[3] advocated cortical uncapping to allow abscess extrusion. Dandy[4] in 1926 recommended simple aspiration as a method of treating chronic abscess formations and as late as 1945[5] was still inclined to "do the least possible." In 1928 Sargent[6] reported five encapsulated abscesses removed *in toto*. Coleman[6] in 1929 reported twelve recoveries in a total of fourteen patients treated by drainage, "using the eye-end of a small rubber catheter." In 1937 Vincent and Askenasy[7] reported their successes in total extirpations, resorting in some to osteoplastic decompression without opening the dura. Tapping the abscess was sometimes necessary to age the lesion for later enucleation. The abscesses were removed intact. Grant[8]

in 1941, listing a bibliography of 207 references, so thoroughly covered the subject of brain abscesses in his collective review as to leave nothing but to quote his closing opinion: "Selected case series show a relatively satisfactory mortality rate, if neurosurgeons or otological consideration were taken of every case admitted to a clinic in which a final diagnosis of brain abscess was made by operation or autopsy, whether the abscess was acute or chronic, adjacent or metastatic, solitary or multiple, and regardless of complications or the patient's condition or admission, the average mortality rate would be about 40 per cent." In 1945 the author[9] reported five cases of total extirpation of abscess lesions, all of which had been totally removed either piece-meal or by aspiration at the time of craniotomy. One might offer the conclusion from the literature on this subject that the problem of chronic abscess treatment should offer no greater surgical mortality than that of surgery of intrinsic cerebral tumors and totally removed abscesses, the morbidity should be no greater than that following the total removal of a benign tumor similarly located.

SYMPTOMS

The only symptom that is constant in a brain abscess is headache. Besides this complaint, there is no classical clinical picture that characterizes this disease. The onset may be insidious and slowly progressive as to suggest the development of a brain tumor. A traumatic history, antedating the onset of headaches, may have symptoms of a chronic subdural hematoma. An acute onset and the rapidity with which neurologic symptoms develop may be typical of a cerebral vascular accident. Other acute conditions which an abscess of the brain may simulate vary from that of an acute encephalitis to intoxications of a chemical or systemic character. An illustration of this group was one patient who found relief for his headaches by the mass use of a proprietary headache powder which contained sodium bromide and acetanilide.

Not until he developed a papillitis was he suspected of having more than a bromide poisoning, the diagnosis of which had been established from blood bromide studies. In the presence of a febrile illness, rapid in the development of neurologic symptoms and with an increased cell count in the spinal fluid, a diagnosis of acute encephalitis is readily understandable.

The headaches as described by the patients in this series were in many instances of localizing value. The intensity of the distress was to the side of the lesion in the majority of the hemispherically located abscesses. Those lesions within the frontal lobes produced distress, focal to the side in which the abscess was located, but on initial questioning the patients complained of headaches across the forehead area. Many patients with an acute onset described their symptom as being a severe pain topographic to the site of the subsequently demonstrated locus of their abscess. Abscesses located in the cerebellum characteristically had suboccipital headaches and the majority described befrontal distress comparable in intensity to the occipital suffering. Except for the patients with occipital lobe abscesses or those patients with meningitis, none of the patients with cerebral lesions complained of headaches below the nuchal line. Unlike the headaches of brain tumors, the headaches in the majority of these abscess patients was not particularly more severe in the early morning hours of the day. Vomiting was a very common accompaniment of these headaches but was not always projectile in character.

If there is a second characterizing clinical feature of a brain abscess, it is a diminution in sensibility. This may vary from an increase in somnolence, to stupor, to profound coma and finally unconsciousness. The old concept of stupor, slow pulse rate and choked disks as being characteristic of a brain abscess had some clinical foundation but it is to be remembered that any acutely expanding intracranial lesion may result in this pathophysiologic cerebral re-

sponse. The degree of reduced sensibility of any patient with a brain abscess depends on the location of the lesion, the extensiveness of the diseased process, the degree of increased intracerebral pressure that is produced by the disease and the rapidity with which the pathologic condition develops. In general, the more acute the process, the lesser the degree of receptivity.

The subjective and objective symptoms of brain abscesses, aside from the aforementioned ones, depend on the locations of these lesions. A frontally placed abscess usually secondary to a sinus infection, a fracture through one of these sinuses or a penetrating wound in this area may exhibit no localizing handicaps. Others may suffer memory defects, personality changes, motor speech disturbances or indifferences. This last unconcern may be for personal attire, business or family responsibilities or sphincter control. Temporally located abscesses are more subject to generalized convulsive seizures than those located elsewhere in the cerebrum. Visual field defects of a homonomous character are to be found objectively and in right handed persons variable aphasic disturbances are common. Acute abscess formation in the motor or sensory cortex is not likely to exhibit focal convulsions and the illness may be ushered in with an abrupt rapid hemiplegia. This abruptness may be such as to simulate a cerebral vascular accident and fundamentally this may be basically such an accident. The insultant is an infected embolus that becomes located in the middle cerebral artery or its branches. Abscess development within the occipital lobes was uncommon in this series and was secondary to compounded head injuries or to osteomyelitis of the occipital bone. The focal symptoms in one such case were those of visual range defects. It would appear that the cortical responses in brain abscesses are those of a destructive nature and not of an irritative character. Cerebellar abscesses exhibit in general symptoms of reliable localizing value—these being nystagmus, ataxia and incoordination of motor ac-

tivities; like cerebellar tumors, they most constantly exhibit choking of the optic nerve heads. Of seven patients with cerebellar abscesses, six had such pathologic changes in their optic fundi. One did not; this particular patient had a complete hemiplegia of a flaccid type and no objective evidences of a cerebellar lesion.

The degree of increased intracerebral or intracranial pressure in a brain abscess is as variable as are the clinical pictures. There were twelve patients in this series who had no opthalmoscopic evidence of intracranial pressure increase. One is apt to see the highest degree of swelling of the optic nerve heads with the most extensive retinal hemorrhages and exudative deposits in patients with increased intracranial pressure due to an abscess. In such a patient these fundic changes alone would deter any risks from a spinal puncture. In a few patients the optic nerves were visualized as being normal. The spinal fluid pressure in such a patient may be somewhat elevated but a normal pressure does not exclude an abscess. The variation of the degree of intracerebral pressure is often more impressively demonstrated at the time of craniotomy. This pressure can be the most alarming factor that the neurosurgeon encounters on dural reflection; the only other comparable pressure increase experienced in the operating room is that which one sees in metastatic disease. This high degree of increased pressure is not only an important factor to be kept in mind at the time of the craniotomy but it is more important that one be cognizant of this tension when one undertakes a ventricular estimation or an air injection for localization of the lesion. If an abscess is suspected before instituting these preliminary studies, one should be alert to the possibility of a surgical emergency. When clinical localization is impossible, in spite of an acute risk, accurate information for effective drainage is the choice. A chronic, well encapsulated abscess may have no greater increased intracranial pressure than a similarly sized benign tumor and an air study or ventric-

ular estimation on such an abscess would entail no greater risk than such a study would for tumor localization. In an acute fulminating abscess a simple ventricular drainage may invite a tragic medullary failure.

The most alarming clinical developments can occur in the shortest periods of time in acute abscess formations. The onset may be as abrupt as a cerebral vascular embolic or thrombotic process. This onset occurs with a headache and extensive incapacitating neurologic symptoms appear promptly. If these objective evidences are a little slow in developing, an intracerebral hemorrhage may be suspected. Temperature elevations over and above the elevations of most vascular cerebral insults may be the only clue to a brain abscess possibility. The optic nerve heads may remain unchanged in the beginning of these illnesses and this absence of choked disks serve only to mislead in the diagnosis. Spinal fluid pressures are at a high normal or above. A frank increase in the cells of the spinal fluid is indicative of the infectious activity adjacent to the meninges. This increased cell count with an elevation in the spinal fluid pressure has been the most valuable diagnostic aid in suspecting these acute conditions. Mortality rates in brain abscesses in the future are to be in ratio to the number of these acute "pus containing, diffuse, infective encephalitics" found in any series.

Historic information in years past was likely to be of more diagnostic value than the neurologic examination. An acute otitis media that ceased draining abruptly or one that was complicated by a mastoid infection necessitating a mastoidectomy, was a common lead for a temporal lobe or a cerebellar abscess. A frontal sinus infection, simple or complicated by an osteomyelitis of the frontal bone, was a definite sign of frontal abscess. A history of a penetrating wound of the skull or a story of a skull fracture through the mastoid or the nasal accessory sinuses with a subsequent development of headaches and a febrile course

was information sufficient to justify suspicion of an intracerebral infection. Any of these patients today have been administered antibiotics, many of these before any clinical manifestations of infection; the great majority of them have had treatment only sufficient to allay the acute process and allow the more serious sequelae to proceed in their chronic development. Maybe "a little help is better than none" in such a serious potentiality but continued and protracted prophylaxis would be more intelligent therapy. The indiscriminate or minimal use of sulfa and penicillin has clouded more than one of what might have been an early human disaster or a good chronologic medical story of an abscess in the brain. In two autopsies in our series it was concluded that the foci which gave origin to the abscess formation had been so allayed from penicillin therapy that in neither instance was there clinical subjective or objective suspicion and the low-grade chronic process of these infections was only discovered at autopsy. These two experiences are such as to indicate x-ray studies of the mastoid bones as well as the paranasal sinuses in abscess suspects or in operative patients with confirmed abscesses who have had any form of antibiotic treatment. Exclusive of the violently acute abscesses of the brain, a clinical temperature chart today is of no value in the diagnosis of these lesions and can serve as a false safety sign to steer one to other diagnostic possibilities. Should this lead one to a craniotomy in a "tumor suspect," the risks, complications and sequelae with intact or piecemeal total resection of the abscess are not what they once were.

TREATMENT

The initial surgical principle in the treatment of a brain abscess is the clearance of the focus from which the abscess had its development. Whether this is with the protracted use of chemotherapy or antibiotic agents or through radical surgical extirpation of this focus or a combination of both is a problem for individual surgical

judgment. If there is a diseased process that gave birth to the brain abscess and this focus is not eradicated, the percentage of abscess cures is materially lessened. Whether the extracerebral pathologic condition is cared for before, during, after or in conjunction with the brain abscess surgery, is again a problem of individual selection. The important fact is to remember that the anti-infectious agents are important adjuncts in the treatment of these infections but are not always to be relied upon to effect a cure, even though the symptomatology might suggest such.

The second surgical principle upon which the successful treatment of a brain abscess depends is accuracy of localization. This is most important, irrespective of the particular type of surgical procedure to be instituted and in spite of the histopathologic phase of the abscess formation. Neurologic, routine radiologic and physical findings may permit precise information as to the site of an abscess. If, however, there is any question of doubt, pneumoventriculography should be instituted. This procedure, as has already been alluded to, may in itself become a very formidable undertaking in certain infectious processes but even in these cases the value of the procedure outweighs its risks, for if the degree of intracranial pathology has attained this serious a level, multiple trephinings and "needling" for the diseased area is a more hazardous procedure than a routine ventricular air study. During the act of localizing the pathologic activity one must decide and plan the surgical attack in keeping with the patient's general condition and duration of his illness; this attack will be influenced mostly by the surgeon's past experiences.

Today, even with a strongly suspected diagnosis of a brain abscess, before any surgical procedure the patient should be well fortified with systemic chemotherapy. If the problem is an acute one, intravenous medication to effect a satisfactory blood level of the sulfa or penicillin, either or both, is the choice of administration. The dosages of these drugs should be of heroic and not homeopathic proportions; in this stage of abscess pathology, the infective organism can be contacted by systemic medication. The question of how protracted this period of treatment shall be is not too difficult to solve in the violently ill patient whose clinical course has been a very rapid one. If such treatment is effective, improvement is likely within twenty-four to forty-eight hours. If these antibiotics are not dramatically beneficial, an increase in the intracranial pressure signs and further reduction in sensibility are the most reliable clinical evidences of a continued, uninfluenced pathologic process. In this acute abscess situation, even surgery is likely to be ineffective but this fact should not influence one against operation; again, "judgment is imperative, time is a factor, and action necessary" if the greater number of these patients are to be salvaged. Decompression alone in our hands has been of no benefit in these patients. Combined with aspiration, a decompression, when the clinical symptoms are more those of increased intracranial pressure rather than sepsis, will be more effective than simple trephine aspiration. It is to be kept in mind that the acute conditions now under discussion are not those in which pus formation is likely to occur. Death in these people is due to the rapid increase in the intracranial pressure and tissue damages incompatible with central nervous system functions. Perhaps in these liquefying cellular destructive phases of the infectious activity, more radical exposures and surgical suction removal of the most evident pathology with the topical use of sulfa and penicillin may prove to be more effective therapy than the conservative efforts of the past. A radical venture no doubt, but certainly the treatment of these fulminating lesions heretofore has been futile. One should utilize one's neurophysiologic knowledge and plan for minor risks of leaving a patient minimally handicapped by such radical efforts.

The treatment of chronic abscess of the brain is easy compared with the treatment

of the acute lesions. When is an abscess chronic? Grossly, when liquefaction and pus formation have occurred, may be a practical answer. The clinical age answer may be one of dispute but certainly a lesion of ten to fourteen days is chronic and beyond this period there are no scientific supports for acuteness other than didactic subdivisions, admitting that within this period acute developments of a secondary vascular character may ensue. As has already been stated these patients are or should be systemically well fortified with sulfa and penicillin therapy. The problem is, "How well is the patient"? If pressure symptoms are subsiding, the septic course suppressed and focal signs are not progressive, these medical therapeutic efforts should not be discontinued in less than eight to ten weeks. In short, as long as the patient is improving no surgical procedures should be instituted. On the other hand, one should not be lulled into a state of false security and rely solely on the antibiotics for a cure of a chronic abscess for there are fewer such cures among chronic abscesses than there are surgical failures. With a total reduction in the occurrence of the disease, it would require a compilation of of many surgeons' experiences to prove this suggestion.

If in a given case of a chronic abscess pressure symptoms demand that one should interfere surgically, a craniotomy should be planned. This should be in accordance with ventricular air depictions of the location of the lesion. A trephine of the skull should be made and a ventricular needle should be introduced in the direction of the already delineated space occupying deformity. If there is a rubbery resistance and it requires some force to introduce this exploratory cannula into this oppugnancy, one has two alternatives. These should be evaluated in terms of duration of disease and the condition of the patient. If the duration of the disease is relatively short and pressure symptoms are dangerous, the osteoplastic efforts of the craniotomy may be performed and in accordance with Vin-

cent and Askenasy's[7] experiences, the abscess may be tapped, the pus evacuated (and penicillin injected) and the surgery discontinued at this place. If pressure symptoms are not too severe, the clinical chart is indicative of no active infectious process and the capsule is profoundly resistant to puncture, the dura may be opened, cortical incision or sacrifice in accordance with the location of the craniotomy exposure and the abscess *in toto*, subsequent to drainage or in piecemeal, may be removed. The technical details of this approach have been previously described.[9]

It is rather likely that King[3] in his cortical marsupialization efforts would not subscribe to his own methods today as a procedure in the treatment of brain abscesses. If the lesion were circumscribed such as to "wander to the surface" after a cortical uncapping, he would likely effect a total extirpation. With effective prophylactic agents against the spread of meningeal infection, it is doubtful that Dowman[2] would be interested today in the production of meningeal adhesions before a surgical attack on an abscess except to allow for chronicity. Coleman, a conservative, having had a string of excellent results from simple drainage, is likely to adhere to the procedures which in his hands have netted the best results. These contributions should be given due evaluation and there are still abscesses, like Macewen's patients, which can be drained through the tract of development with complete results. The choice of treatment for an uncomplicated convalescence and minimal morbidities in the experiences of the author are those abscesses which have been extirpated at the time of craniotomy.

SUMMARY

Antibiotic and chemotherapeutic procedures have tremendously reduced the incidence of brain abscesses and this therapy is indicated irrespective of surgical procedures instituted. This form of therapy in its prophylactic efforts has served in

more than one experience to chronicize and deter diagnostic concepts.

The occurrence of brain abscess formation is still common enough to command the interest of all concerned.

There is no clinical picture that characterizes this disease.

Eradication of the focus of origin of a brain abscess is the first principle in the surgical treatment of these lesions.

Accurate localization of the abscess process is the second most important surgical consideration—a procedure at times not without grave risk.

Total exclusion of the abscess is the surgical goal of treatment and radical efforts to date have been the most effective in this accomplishment.

No cure of a brain abscess should be ascribed in any patient without benefit of either a total surgical extirpation of the lesion or a confirmation of the extermination of the disease by pneumoencephalographic studies.

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DIAGNOSIS OF INTRACRANIAL ANEURYSMS*

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INTRACRANIAL aneurysms are frequent causes of sudden, severe intracranial symptoms. Certainly, the incidence of intracranial aneurysms is much higher than has been suspected in the past. Many patients who succumb from a so-called cerebrovascular accident actually die of a ruptured intracranial aneurysm. Intracerebral hemorrhage with ruptured aneurysms occurs frequently, that is if the aneurysm involves one of the branches of the middle, posterior or anterior cerebral arteries. Aneurysms that actually involve the circle of Willis manifest their rupture by evidence of subarachnoid hemorrhage rather than intracerebral hemorrhage. The incidence of subarachnoid hemorrhage is greater than that of intracerebral hemorrhage because of the anatomic relationship of the larger arteries to the brain. All the larger branches actually lie in the subarachnoid space. Rarely do aneurysms occur in the minute vessels that penetrate the brain substance.

In a personal experience with 114 intracranial aneurysms in 110 patients, the etiology presumably was related to congenital defects in the arterial walls. One exception was a patient with mycotic emboli. Hypertension was noted in twenty-three patients. In none was syphilis a factor.

It is of interest that aneurysms were found in seventy-two females and thirty-eight males. Forty-seven aneurysms were found on the right and sixty-three on the left side.

Unfortunately, there is no clinical syndrome that makes the diagnosis of an unruptured intracranial aneurysm a reasonable certainty. Without evidence of subarachnoid hemorrhage, however, there are certain symptoms which should arouse suspicion of its presence.

That the diagnosis of intracranial aneurysm should be made, if at all possible, before rupture, is dramatically presented by Magee and also more recently by Hamby. Magee found that fifty-two of 150 patients with subarachnoid hemorrhage died in the primary attack. In fifty of the ninety-eighty survivors of the first attack, recurrence took place and thirty-two patients failed to recover. In other words, 35 per cent died in the first attack and 21 per cent succumbed in recurrent attacks. Of the 44 per cent who survived, many were either partially or totally disabled as a result of the cerebrovascular changes. Hamby, in an excellent survey of 130 verified intracranial aneurysms found at the Buffalo General Hospital since its beginning, including autopsy material, reported 39.9 per cent died in the first attack and 28.5 per cent died of recurrent attacks.

Since many of the recurrent hemorrhages take place within three weeks following the initial hemorrhage, it is important that an attempt is made to establish the diagnosis as well as to determine whether or not the aneurysm is located at a site that is amenable to surgical intervention, or whether the patient's chances are as favorable without surgery as with surgery.

The whole problem from the practical standpoint depends entirely on the question as to whether an aneurysm is definitely the cause of the sudden intracranial symptoms. If so, what should or can be done about it? The statistics of Magee, as well as those of Hamby, emphasize the seriousness of the condition. The initial symptom

of many of these patients was the result of the rupture manifesting itself by a subarachnoid hemorrhage, the patient being normal in every way subjectively before the rupture. There is no possible practical means that one could utilize to uncover an unruptured aneurysm in those patients in whom no previous symptoms were present. Therefore, it is important that patients with symptoms that might indicate an aneurysm should be investigated.

In the surgical experience with 110 patients in whom intracranial aneurysms were verified, the most striking feature was that only nine patients with intracranial aneurysms were encountered in a period of ten years and 101 were found in the past four years, mainly because of an awareness that such a condition might be present. Because of that suspicion, diagnostic aids were utilized either to prove or disprove the presence of an aneurysm. That Dandy had a similar experience is indicated in his monograph on 108 cases of aneurysms which covered hospital reports for a period of fifty years; 40 per cent were necropsy reports and 60 per cent personal surgical experience in twenty years. Thirty-six of these patients were operated on in a period of six and one-half years after his interest had been stimulated in the surgical treatment of aneurysms.

The initial symptoms other than that of subarachnoid hemorrhage in many instances were bouts of unilateral headaches and in others, periodic attacks of generalized headaches. In only four of the 110 patients were typical unilateral attacks of migraine the presenting complaint. Eighteen of the patients had no symptoms previous to the actual rupture. Eight patients had atypical facial neuralgia. Seven patients noted diminished vision as a presenting symptom. In twelve patients the symptoms were initiated by generalized convulsions. Eighty-two of the patients had a history of previous subarachnoid hemorrhage. The symptoms of subarachnoid bleeding may be confused with those of meningitis because of the stiffness of the neck, generalized headache and elevation of temperature. Therefore, one must be alert in taking the patient's previous history to be certain that a diagnosis of previous meningitis might not actually have been subarachnoid bleeding. Forty-five of the patients had a single subarachnoid hemorrhage; the remaining eighty-two patients had two to eight previous hemorrhages. In eighteen patients the symptoms were of gradual onset and were usually caused by local pressure from a large aneurysm in the region of the sella turcica.

Sudden hemiplegia occured in eleven patients. This development in a patient with aneurysm is either due to intracerebral hemorrhage, propagation of thrombosis or an embolus which has broken off the thrombus in an aneurysm.

Periods of dizziness were noted in a sufficient number of patients to justify its mention. In none of these patients was vertigo the only symptom.

Ophthalmoplegia is probably the most common finding in patients with large aneurysms involving the internal carotid artery or in patients with extensive hemorrhage along the third cranial nerve. The latter is especially vulnerable to sudden localized pressure. Since this is true, one cannot be certain as to the exact location or the size of the aneurysm. It is a valuable clue, however, as to the side of the circle of Willis that it involves. Sixth nerve palsy occurred in six of the 110 patients. These palsies also were on the aneurysm side of the circle of Willis, and associated with aneurysms of various sizes with no uniform pattern as to location. Papilledema was found in eight patients; one was associated with subretinal blebs of hemorrhage, pathognomic of subarachnoid hemorrhage. Nineteen patients had optic pallor owing to direct pressure of the aneurysm on the optic nerve.

Involvement of the fifth cranial nerve was present in the large subclinoidal aneurysms, with pressure directly on the first and second divisions. Manifestation of pain in the first and second division of the

fifth cranial nerve was frequently the initial symptom of ruptured aneurysm. The seventh cranial nerve was not involved in any of our patients except for central facial weakness. The functions of the eighth, ninth, tenth, eleventh and twelfth cranial nerves were changed from normal in a patient with a large aneurysm of the basilar artery. In this patient the ninth cranial nerve pressure was indicated by a typical glossopharyngeal tic douloureux.

The usual cause of a bruit in aneurysm is an arteriovenous opening which occurs most frequently by rupture of a subclinoidal aneurysm of the internal carotid artery into the posterolateral surface of the cavernous sinus. The incidence of bruit in aneurysms is low; however, it can occur in patients with localized atherosclerotic changes in the internal carotid artery which have narrowed the lumen of the artery locally. That this can and does occur has been demonstrated in several instances. Partial occlusion of the internal carotid artery in the neck is an example of localized narrowing of an artery producing a bruit or thrill in that region. In seventeen of the 110 patients a distinct bruit was present because of a venous communication with the cavernous sinus. In two patients a direct communication of a branch of the anterior choroidal artery with the vein of Galen caused bruit. Another bruit was elicited in which partial thrombosis had occurred in a large aneurysm of the third portion of the internal carotid artery. In three patients bruit was elicited which could temporarily be occluded by compression of the internal artery. No changes in the branches of the carotid system could be noted by arteriogram, however, on the side on which the bruit was elicited. Two of these patients had aneurysms involving the vertebral artery. In one patient visualization of the vertebral artery and internal carotid artery was normal. Ligation of the internal carotid artery changed the character of the bruit and completely abolished it for a period of a few hours; however, it then returned. The cause of the bruit, therefore, is undetermined.

In eliciting the history of a patient with a suspected intracranial aneurysm, direct inquiry must be made as to whether the noise he complains of is localized. Also, is it synchronous with his heart beat and does its character change with position? If a bruit can be heard with a stethoscope the internal carotid artery may be occluded by digital compression on the side of the bruit. If the bruit disappears completely by occlusion of one internal carotid artery, it is quite certain that it involves the carotid system and not the vertebral arterial system. If the patient complains of a cranial noise synchronous with his pulse which cannot be heard with the naked ear or the stethoscope, a stethotron should be utilized. If this fails to corroborate the presence of a bruit, the internal carotid arteries are alternately compressed. If there is no subjective reduction or cessation of the noise, one can be reasonably certain that the bruit does not involve the carotid system. I know of no method in which a bruit of the vertebral system can be eliminated diagnostically without direct exposure of the artery in the neck unless the artery enters the vertebral foramina higher than usual and possible occlusion is made by digital compression low in the neck.

In any bruit that can be heard equally well over the entire skull which is not due to aneurysm, one must consider advanced Paget's disease or extensive blood vessel anomalies. The presence of a bruit intracranially which occurs suddenly with an association of pulsating exophthalmus means that there is a communication of a large artery with the cavernous sinus unless there is a history of a penetrating injury, such as gunshot and stiletto wounds, or a basilar skull fracture. The fistula between the artery and vein is the result of a rupture of an aneurysm into the cavernous sinus. This occurrence, of course, is lifesaving since it is far safer for an aneurysm to rupture into the cavernous sinus than into the subarachnoid space.

The typical history of a ruptured aneurysm of the anterior two-thirds of the circle of Willis is manifested by sudden onset of a severe unilateral frontal headache, extending rapidly posteriorly, becoming generalized, followed by stupor for a varying period of time—from several hours to two or three days. When the patient regains consciousness, drooping of the eyelid on the affected side is noted with a partial or complete ophthalmoplegia. The generalized headache and stiffness of the neck persists for several days or weeks. Bloody spinal fluid, of course, verifies the diagnosis of a ruptured intracranial aneurysm. It does not, however, indicate the exact site or determine whether or not the aneurysm should be attacked intracranially or indirectly.

From the standpoint of treatment, the importance of verification of the diagnosis cannot be overemphasized so that one is in the position to advise the patient and family intelligently. As to the treatment, the surgeon must know the exact site of the aneurysm; he must know the tolerance of each cerebral hemisphere to reduction in blood supply (collateral circulation); he must know the risk involved in non-surgical treatment versus indirect or direct surgical intervention; he must know statistics as to the time a secondary hemorrhage is likely to occur; he must know whether the aneurysm is located where its removal is likely to cause permanent hemiplegia. For this reason, arteriography[3] plays a most important part in the problem of aneurysms. Arteriograms in most instances are imperative both from the standpoint of the presence of an aneurysm as well as to determine its exact location, allowing one to judge from previous experiences as to whether its location is relatively safe for direct intracranial attack by excision or trapping or whether the indirect method by proximal ligation in the neck would be the safer of the two procedures.

The plain stereoscopic x-rays may give valuable information as to the presence of

FIG. 1. Sizable subclinoidal aneurysms have a tendency to erode the anterior clinoid upward or sharpen it on the inferior border. Since the majority of aneurysms are in the region of the circle of Willis, the roentgenograms should be closely scrutinized in the region of the sella turcica for calcification.

aneurysm. (Fig. 1.) Only the large aneurysm which is in direct contact with bone can possibly cause changes with the exception of possible calcification which may occur in an aneurysm. It is usually manifested by a fine crescentic outline or dense calcification (Sosman and Vogt). Thinning of the lateral margin of the optic foramen has been described by Jefferson.

The final diagnosis, therefore, rests on, either the arteriogram or direct surgical exploration. In my experience, arteriography has been of great help, allowing one to study the relationship of the aneurysm to the main branches of the circle of Willis. For that reason, arteriograms are now made in all patients suspected of having an aneurysm. Over 200 arteriograms have been made in most cases with thorotrast; however, more recently diodrast has been employed with no untoward results.

The question arises, when should arteriograms be instituted in patients with recent subarachnoid hemorrhage? I have no

FIG. 2. Equipment necessary for injection of carotid artery by the indirect method.

hesitation in injecting thorotrast or diodrast at any time if a No. 18 gauge needle is used since any increased intra-arterial pressure caused by the injection would be dissipated before reaching the site of the aneurysm. Many times it is impossible to determine from the physical examination or history the side on which the aneurysm is situated; therefore, both carotid arteries and their branches should be visualized by the closed method so that the proper site of the leaking aneurysm may be determined before surgical interference is instituted.

The ligation of an internal carotid artery can be of no value in an aneurysm which does not involve the anterior two-thirds of the circle of Willis. Therefore, in a non-localized bleeding aneurysm, if no aneurysm is disclosed by bilateral carotid arteriography, the vertebral arteries with their branches should be visualized.

The choice of the contrast media should be made on the basis of whether the patient is sensitive to iodine. At the present time I use 35 per cent diodrast in all patients who show no evidence of reaction to a test dose of diodrast. Thorotrast is used whenever there is any question of sensitivity to iodine. Diodrast is preferable, even though the contrast is not as sharp as with thorotrast since the injection may be repeated several times if for some reason the arteriograms are unsatisfactory, whereas 20 cc. of the thorotrast is about the maximum volume one should use in any given patient, and preferably 10 cc., because it is not excreted.

The method of injection is, of course, important. At present I use only the closed method since it may be employed in patients with equivocal signs of aneurysm in whom the air studies are normal. Arteriograms can be made with the patient still asleep from pentothal anesthesia, immediately following the review of the encephalogram. In patients with evident manifestations of aneurysm one is perfectly justified in making a direct exposure for the arteriograms. The reason I do not use the open method routinely is that one is inclined to ligate the internal carotid artery in the neck as soon as the aneurysm can be seen on the wet x-ray film. Adequate study may not be made of the exact site as to its relationship and involvement of other branches of the circle of Willis. If a direct attack seems indicated, one should allow as much blood supply to remain as possible, and therefore ligation in the neck is left undone. In other words a more deliberate plan of treatment may be followed. With normal air studies, vascular changes have not been excluded. For that reason the indirect or the so-called closed method of arteriography is an added minor procedure, whereas exposure of both carotid arteries verges on the major, nor is one assured of better filling with the open than with the closed method. Incidentally, many more aneurysms will be uncovered by performing an increased number of arteriograms. I am, therefore, convinced that the combination of encephalograms and arteriograms should be used more frequently than at present; that is, if the encephalogram shows no gross changes, this procedure may immediately be followed by the closed

FIG. 3. Relative position of patient and operators to the x-ray equipment.
FIG. 4. Position of patient on table to prevent overextension of neck during injection.

method of arteriography under the same anesthetic agent.

The technic of arteriography is simple, even though it took me a long time to develop a method that produced satisfactory arteriograms. It is important to keep in mind that the completion of a satisfactory arteriogram depends on the teamwork of the operator, the x-ray technician and, of course, the physical equipment, namely, the x-ray apparatus and the syringe and needle which must be in perfect working order. (Fig. 2.)

The patient is allowed to remain on the carrier stretcher on which he has been transported to the x-ray department. (Fig. 3.) The wheels of the carriage are firmly fixed so that no motion can take place at the time of the injection. With the patient on his back, a pillow or folded blanket is placed underneath his shoulder blades and two folded Turkish bath towels are placed under the occiput. Care must be exercised to prevent overextension of the patient's neck, either by lifting up the chin during the pentothal

anesthesia or by allowing the head to rest considerably lower than the chest. (Fig. 4.) It would seem probable that, by extension, the large vessels of the neck would be held on a stretch, thus allowing the needle to enter the lumen more readily.
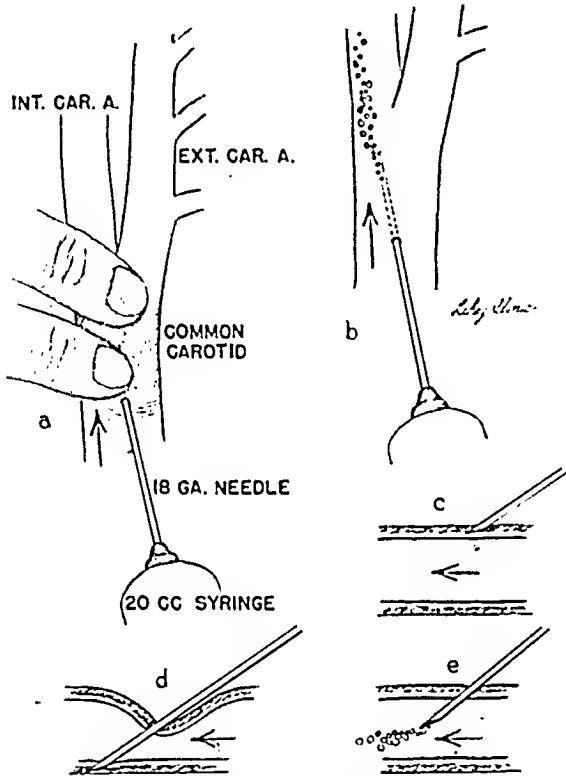
FIG. 5. a, Position of left middle and index fingers; the middle finger is over the carotid bulb; the index finger is over the common carotid artery. The point of the needle engages the common carotid immediately beneath the index finger. b, Demonstrates "streamline flow" of the contrast medium. c, Sharp-pointed, curved, beveled needle engaging adventitia. d, Needle withdrawn slowly so that it is well in the lumen of the artery. e, Compression of arterial wall by the needle, indicating the reason arterial blood may not enter needle.

The opposite, however, has been my experience. The lumen of the artery is actually narrowed and may in some instances be occluded by the stretch over the tubercle of the sixth cervical vertebra. The patient's head is allowed to remain in a horizontal plane. The index and middle finger of the left hand are used to palpate the common carotid artery, the middle finger (Fig. 5) is allowed to rest on the carotid bulb and the index finger on the common carotid artery beneath.

A No. 18 gauge needle, 2 inches long, with a very sharp point and moderately curved bevel, attached to a 20 cc. Luer-Lok syringe by a two-way petcock, is directed so that the point of the needle engages the adventitia of the common carotid artery immediately beneath the site of the index finger. Delicate pressure is then exerted on the needle with the syringe, and immediately released if there is any tendency for the artery to roll to one side, since this means that the point of the needle is engaged eccentrically and not dead center. When one is certain that the needle is dead center over the artery, which must be noted entirely by the feel of the index finger of the left hand and the transmitted feel of the needle through the syringe to the right hand, the needle may then be inserted into the artery, arterial blood can be seen pulsating into the citrate solution which is in the syringe, and it has a tendency to push the piston of the syringe outward. If arterial blood does not enter the syringe, the needle is slowly withdrawn, since many times the arterial wall has become constricted at the site of entrance by the pressure of the needle and, as one withdraws the needle slowly, the artery expands and arterial blood may be seen pulsating into the syringe. By careful manipulation one can then place it well into the artery. The next step is to be certain that the needle is well engaged in the lumen of the artery and will not be displaced by the injection. It is well to take several practice injections with the citrate solution which is used to prevent clotting of blood in the syringe and needle. If several cubic centimeters can be injected rapidly and a free back flow of arterial blood takes place, with the needle directed toward the lateral wall of the common carotid artery, the syringe with the diodrast is then attached and the solution injected as rapidly as permitted through the No. 18 gauge needle. Great care must be exercised to prevent bubbles of air, which become caught in the hub of the needle as the

syringe is attached, from being injected into the carotid system. To prevent this air from being injected, blood can be allowed to flow into the contrast medium so that the two or three small bubbles of air will be carried to the upper portion of the syringe and remain there during the injection. Certain serious sequelae can take place from air emboli and may account for reported convulsions occurring during injection.

It is important to inject the contrast medium toward the lateral lumen of the common carotid artery because a definite law of "streamline flow" holds true in the arterial system. In other words, if the contrast medium is injected laterally and downward, it will follow the blood stream into the internal carotid artery; whereas if the contrast medium is injected medially and upward, most of it will appear in the external carotid artery.

It is necessary that the x-ray technician be made aware that the injection is about to start. The x-ray tube is prepared for making the exposure. The head is thoroughly aligned with the upright automatic Bucky, suitable for stereoscopic exposures, which is used routinely for chest roentgenograms, 20 cc. of the dye has been placed in a 20 cc. syringe and 15 cc. is injected rapidly, at which time the first exposure is made. Then the injection is continued during the automatic shifting of the x-ray tube and the films for the second exposure. This allows adequate filling for stereoscopic films and in most instances the latter are more satisfactory than a single lateral view and an anteroposterior view. (Fig. 6.)

A word of caution should be inserted here that the operator and assistant must be careful not to allow themselves to be exposed to x-rays beyond the dosage that is safe for their own welfare. The dosage that can be tolerated may readily be computed by the roentgenologist and one should abide by his judgment.

Aneurysms of the posterior third of the circle of Willis fortunately occur in only

FIG. 6. Arteriogram with aneurysm involving a vestigial artery, the rupture of which caused complete third nerve paralysis. The anteroposterior arrow points to the internal carotid artery, the posteroanterior arrow to the aneurysm. The arteriograms taken stereoscopically afford an excellent method of studying the relationship of the aneurysm to the main arterial branches.

25 per cent of all aneurysms involving the intracranial cavity and can be visualized by arteriography of the vertebral arterial system. Unfortunately, the closed method of injecting the contrast medium is more difficult; however, it can be executed in individuals with a high subclavian arterial arch. Direct exposure of the vertebral artery can be done. The law of streamline flow in the vertebral system is of importance in that the opaque medium follows the blood flow from the vertebral artery into a common reservoir, the basilar artery, filling only the branches on the side injected as well as the posterior cerebral artery on that side.

COMMENT

The diagnosis of intracranial aneurysms should be made before intracranial catastrophe takes place if at all possible.

An awareness of the frequent occurrence of intracranial aneurysms and of the fact that many of these patients may be helped surgically should stimulate us to institute diagnostic means that may reveal the aneurysm before actual rupture occurs.

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FOLLOWING trauma to the head intracranial bleeding of four different types may occur. Three types involve the brain only indirectly by pressure or irritation. When an extradural hemorrhage is suspected, an exploratory burr hole should be made in the mid-portion of the temporal bone just below the insertion of the temporal muscle. The skin incision should be outlined so that the initial incision can be converted into a subtemporal decompression.

INTRACTABLE PAIN DUE TO CANCER

TREATMENT BY NEUROSURGICAL METHODS

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NATURE OF PAIN DUE TO CANCER

STRANGELY enough cancer is not inherently a painful lesion. Pain is due to secondary or neighborhood effects. Of chief consideration are obstruction of a viscus, infiltration of nerves, compression of nerve roots or trunks against bony structures, infiltration, tumefaction and swelling in tissues snugly invested by fascias and rich in nerve trunks and plexuses and lastly infection and inflammation. Obstruction at some locus in the gastrointestinal tract is illustrative of the first classification. Carcinoma of the stomach, sigmoid colon and rectum are relatively painless until the symptoms of obstruction supervene. Inasmuch as obstruction is amenable to general surgical procedures, the neurosurgeon will seldom see those patients before metastases or extensive infiltration has occurred. Infiltration of the brachial plexus and nerves in the axilla is common in patients with metastasis from breast cancer. Compression of nerve roots is manifest in metastases to the spine. Metastases to bone are relatively painless unless there is some gross distortion or involvement of nerve roots. Witness the painlessness of metastases to the skull. Metastases to the spine from carcinoma of the uterus, breast and rectum are often painful and one finds bulging and stripping of the dura with crowding of nerve roots at their intervertebral foramina. Seldom do such metastases transgress the dura early to become intradural, a point worthy of mention. Metastases which are late in manifestation and which grow slowly may at first cause pain in a single root distribution.

The presence of a primary carcinoma may not be suspected or the significance of one treated some years before may be overlooked. The following case is illustrative: N. R., a white woman of seventy-one, was admitted to Mercy Hospital in Denver on January 16, 1945. She complained of severe pain in the distribution of the left occipital nerve. Her history revealed that a carcinoma of the breast had been removed five years ago but since careful examination, including roentgenograms of the skull and cervical spine, were negative a diagnosis of occipital neuralgia (inflammatory?) was made. The persistent and severe nature of the pain prompted avulsion of the occipital nerve on the left. This provided some relief for a short time. She then went through a period of vestibular disturbance that was taken to be labyrinthitis and it was not until two years after avulsion of the occipital nerve that involvement of the ninth to the twelfth cranial nerves on the left, with hoarseness and dysphagia, betrayed the true nature of the pathologic condition. A roentgenogram of the skull now revealed punched out areas near the foramen magnum. In a somewhat desperate attempt, at the patient's behest, the nerves were explored. The ninth, half the vagus and the second and third cervical sensory roots were cut. Metastases had grown *en plaque* about these nerves extradural but had not become intradural. During the month that she lived after operation, metastases grew rapidly. Tumefactions raised over the skull with incredible rapidity.

The significant lesson is that the agonizing pain which she suffered for almost two years might have been treated intelligently had the probability of obscure metastases been given due consideration.

The pain notoriously associated with carcinoma of the uterus and uterine cervix is undoubtedly due to infiltration of tissue planes in the pelvis with consequent infiltration of the lumbosacral plexuses and compression against the bony pelvis.

The anatomy of the neck is characterized by closely invested tissues rich in nerve trunks. Tumefaction and induration involving tissues of the neck are characterized by pain and tenderness.

When cancer erodes mucous membrane or skin, infection and inflammation develop and progress deeply into the lymphatics and fascias. Carcinoma in and about the buccal cavity is the outstanding example especially when infiltration involves the throat and neck. As an exception, carcinoma of the lip even with metastases to regional nodes is not always painful.

Hence, from this brief resumé it might be conjectured that neurosurgery is enjoined largely for advanced cases of carcinoma of the uterus and cervix, breast and buccal cavity. Ninety per cent of the author's chordotomies have been performed for pain of carcinoma of the uterine cervix with pelvic infiltration. In the minority group are a few patients with carcinoma of the rectum with metastases to the spine. The remaining chordotomies were performed because of lesions other than cancer.

TREATMENT OTHER THAN NEUROSURGICAL

While it is not entirely within the province of this paper to discuss other methods of treatment, those most commonly employed are mentioned briefly for orientation and because they have such an inextricable bearing on the subject. Toward this end, narcotics, radiation and sex hormones deserve some discussion.

Narcotics. Morphine is naturally the first court of appeal when pain becomes a real and persistent symptom. When pain becomes a symptom of inoperable cancer, it is usually persistent and the attending physician has little else at his disposal but this greatest of all pharmacologic blessings

to meet the exigency and ease the pain of his patient. The administration is continued for varying periods until the inevitable threat of addiction becomes manifest or until the drug becomes inefficacious. The young and enthusiastic neurologic surgeon is likely to underestimate the demanding and natural circumstances under which the drug is administered by the family physician and to place too great an emphasis on the evils of addiction and its contaminating influence on the success of surgery. Not all patients with the cancer-pain syndrome are appropriate candidates for surgery. If a patient, after radiation, can be made relatively comfortable with the drug throughout his limited days, the author knows of no superior treatment. There are exceptions, examples of which are given in this paper, and it is not easy to exercise perfection in judgement in the given case. The "magic drug"* would be the treatment par excellence for it stands to reason that surgery designed to relieve intractable pain only, for one whose time is short, is a major, last resort undertaking and an indication of the dire need for a less trying and less expensive management.

Radiation. It would be presumptive in so short a space to attempt to give due credit to this fascinating branch of medicine in which the energy of electromagnetic waves is enjoined to destroy abnormal flesh and preserve the normal. Of the limited contact and experience of the author, the following convictions have conditioned his judgement and practice:

1. The effects of radiation upon cancer pain are unpredictable but beneficial effects prevail by such a wide margin that an honest effort to use it must be encouraged. When properly administered, harm is so minimal that it can be discounted. The prospective benefits are two-fold: possible relief of pain and attenuation of growth propen-

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* At the time of writing a new drug, "dolophine," is being carefully dispensed under controlled management. It has the analgesic properties of morphine with minimal powers of addiction. This field of research is most intriguing and urgent.

sities of the neoplasm. Relief of pain is often quite satisfactory for a time although unfortunately it is not often permanent. It is, nevertheless, a gain in both time and courage and the administration is in no way an ordeal for the already tired, weary and anxious patient. The immediate relief of pain and quieting effect of radiation on bone sarcoma, for example, has been very impressive to the author.*

If radiation does not cure, it certainly attenuates the new growth to varying degrees and reduces toxicity with systemic improvement. Occasionally, a very happy and remarkable cure ensues. Cooper and Archer,[1] to cite an example, report the case of a six year old child appearing *in extremis* from metastasizing Ewing's tumor of a rib. Half-hearted radiation resulted in a cure. They emphasize that radiation often relieves pain due to bone metastases.

2. The author does not believe that this branch of therapy has reached its limitations and when considering the desperate nature of inoperable cancer what other effort can be better recommended? Perhaps the fact that wave physics and clinical medicine are in practice, such full time and divergent fields has delayed progress that may yet be attained. He also believes that the domain of roentgenology, particularly that of radiation, has been well manned by conscientious and progressive disciples. Gratifying reports are not often overdrawn.

In short, as a matter of policy and with very few exceptions, the patient with inoperable carcinoma and pain should be turned over to the radiologist at least for the benefit of consultation and certainly before undertaking neurologic surgery.

Sex Hormone Therapy. While this subject is in its infancy, it is of sufficient importance to be listed here. Adair[2] reports that after about two weeks of testerone therapy the relief of pain from bone metastases of breast cancer is usually very real. Huggins, quoted by Adair,[2] reported cures as high as 25 per cent in patients with cancer of the prostate. In the discussion of a paper by Crutchfield,[3] Archer states that patients having carcinoma of the prostate associated with pain should have the benefit of orchiectomy. The relief of pain is dramatic in many instances.

NEUROSURGICAL APPROACH

The selection and preparation of patients for neurosurgery should be made with considerable perspective and discrimination. This is said because if neurosurgery is to be effective, it must often assume the proportions of major surgery. Neurological deficits are likely to result as side effects when incisions are made into the mesencephalon, medulla or spinal cord.

As already stated, and except for some special reason, the patient should be a victim of *intractable* pain. Radiation should have been given a fair trial. Neurosurgery should be considered the last court of appeal but it is by no means implied that it should be delayed unnecessarily.

Due consideration should be given to three aspects of the undertaking: the neoplasm, the pain and the patient. In respect to the neoplasm it is almost embarrassing to insist that the diagnosis of inoperable malignancy should be unassailable. Fortunately, mistaken diagnoses are not common because histologic confirmation has become almost universal practice and by the time a cancer is considered inoperable it usually is. Diffuse pain in the pelvis, low back, in the bladder region and with some radiation in the distribution of the lumbosacral plexus on one or both sides,

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* Relief of pain is probably accomplished because radiation quenches inflammation thereby shrinking the mass and relieving tension. This may be the only pain relieving property of x-rays. If so, one might make prophesies in relation to a given lesion and explain the limited duration of relief from pain by radiation. It is probably true that constant and certainly pulsating pain from any tense swelling is due to a stretch of nerve fibers. If tension is diminished, as in the letting of pus, pain abates.

are hardly to be mistaken in respect to their ominous significance if they develop some time after a proven carcinoma of the uterus. The type of neoplasm, its grade of differentiation and other characteristics which help to predict its life history are pertinent. On the whole, a highly undifferentiated cancer that is rapidly growing and taking its toll of the patient in the form of cachexia and mental torpor might be more wisely treated by narcotics. There is no virtue in expediting neurosurgery two weeks before the patient dies. On the contrary, and as a general statement, a scirrhous type of cancer may, with aid of radiation, remain relatively quiescent for several years and if the patient is free of pain show little effect upon the general system. Thus, a young woman (G. B.) was admitted to the University Hospital in Iowa City in December, 1941, with severe intractable pain in the pelvis and lower extremities resulting from inoperable carcinoma of the cervix. Bilateral section of the spinothalamic tracts at the first dorsal segment abolished pain completely. Although she appeared frail and exhausted, we realized that this had been due more to pain than carcinoma. With the will to live, she was discharged in three weeks having gained weight and presenting relatively little untoward neurologic deficits. She joined her husband who was in the service and lived for five years without pain and to all appearances in good health. The break in health was then rapid. The tumor throughout its course was hard and nodular to palpation.

Experience teaches that pain may be overestimated by some patients. Some may be inclined to over-rate the benefits of surgery and not until they are thoroughly apprised of all factors do they conclude that the pain which they have is satisfactorily amenable to simple drug therapy. While it often requires only minutes to be convinced of the realistic and severe nature of pain and the justification for early surgery, there are equivocal situations in which it is well to visit the patient a number of times before coming to a decision. By thus becoming better acquainted with the patient and observing his reactions, the author has forstalled operations that proved better left undone.

Any discussion concerning the patient should be prefaced with the fact that the purpose of the type of surgery under discussion is strictly humanitarian. The objective is not only to eliminate the sensation of pain but to help the patient feel as generally contented as possible. Toward this end the surgeon should become acquainted with him, instilling confidence and establishing rapport. While firmly discussing the possible sequelae of operation, the unpleasant features can be minimized and relative values made clear. These practices have on occasions prepared the patient and allayed anxiety so that he was contented and at peace although the lower extremities were weakened and the bladder atonic. In short, it is poor policy to attempt in any way "to sell" the operation to those patients. When the facts are placed before them with clarity and human rapport, their election should be respected as the best judgement.

OPERATIVE METHODS

In electing some operative procedure to relieve intractable pain, the author has become convinced that the "all or none" policy is best. When the decision is made after proper deliberation, a procedure designed to give optimum and certain results should be carried out. Lesser and "more conservative" procedures or "trials" and multiple operations only torture a weary patient and nullify confidence. The author has concluded, therefore, that except for pain in the face and throat, a section of the spinothalamic tract at an appropriate locus is the procedure of choice at the present writing.

Intraspinal subarachnoid injection of alcohol, various types of sympathectomy and rhizotomy have a very limited application.

Subarachnoid Injection of Alcohol. In my experience the subarachnoid injection

of absolute alcohol has been of little assist-
ance in treating the pain of cancer. This is
because the great majority of patients seek-
ing aid have diffuse pain in the pelvis and
lower extremities, involvement of the bra-
chial plexus or pain in the head and face.
The first two categories are best treated
by other methods than deafferentation
and the third category does not lend itself
to subarachnoid injection of alcohol
for obvious reasons. The use of alcohol is
practically limited to the distribution of
the thoracic nerves and finds wider appli-
cation in the treatment of pain from other
causes than cancer. Disadvantages in its
use may be listed as follows: It is difficult
to obtain the wide zone of analgesia that is
usually necessary. With 1 to 2 cc. of abso-
lute alcohol one may expect to eliminate
the nearest sensory root and partially
eliminate the two adjacent roots. It is
precarious to inject alcohol in the region
of the conus medullaris. The origin of the
lumbosacral nerves is concentrated here
and some paralysis of the lower extremity
associated with paralysis of the bladder
sphincters almost invariably results. To
illustrate the limited but valuable applica-
tion of a method of this kind, I recall a
patient with carcinoma of the bladder and
severe pain referred to the end of the penis.
The patient was placed in the prone posi-
tion with buttocks elevated and 1 cc. of
absolute alcohol injected into the sub-
arachnoid space at the L-5 interspace. The
relief of pain was immediate and perma-
nent and since the bladder sphincters were
already paralyzed this constituted no
hazard.

Rhizotomy. This is the treatment of
choice for pain in the face, tongue, mouth
and throat. Any further application of
deafferentation, as Bronson Ray[4] points
out, is limited to pain in the neck and
trunk. He does, however, recommend
rhizotomy in respect to roots of the bra-
chial plexus and partial rhizotomy in se-
lected patients. Excluding the head, cancer
pain almost always involves the brachial
or lumbosacral plexuses to some extent.

Hence, neurosurgical treatment must take
these territories into consideration. Aside
from the unsatisfactory zone of the shoul-
der and upper extremity I have in recent
years entertained no equivocation in re-
gard to the choice between rhizotomy and
chordotomy for cancer pain.

Sympathectomy. Insofar as I know,
sympathectomy alone has no real applica-
tion in treating cancer pain. Very little is
known about sympathetic pain bearing
afferents except in relation to the viscera.
Even here I use the term *sympathetic affer-
ent* with reservation since many anatomists
still avoid the connotation and prefer the
term *visceral afferent.* The weight of opinion
eases them into the category of somatic
afferents functionally although they do
course through the sympathetic chain. In
any case, whatever the sympathetic factor
may be, it probably never exists alone in
our cancer patient and it is expunged along
with the somatic factor by either an appro-
priate rhizotomy or section of the spino-
thalamic tract. Deference should be given
to one possible exception, that of presacral
neurectomy. This has a limited application
in the treatment of certain kinds of pain
emanating from the uterus and distal por-
tion of the bladder. I have yet to see a case
of cancer pain in which it could with any
reason be proposed as a single procedure.

I have noted a tendency for some
surgeons to regard presacral neurectomy
as a cure-all for pain in the pelvis. Even
though the neurectomy were actually
accomplished through the hazards of a
carcinoma-infested pelvis, it constitutes
only a futile operation.

OPERATIVE PROCEDURES OF CHOICE

By and large and from an operative
standpoint, the neurologic surgeon is called
upon to relieve pain in one of three cor-
poreal territories: (1) The face, mouth,
tongue, throat and neck; (2) the shoulder
and upper extremity and (3) that part of
the body from the level of about D-5 down.
In the first case, surgical elimination of the
transmission of pain has been developed

about as far as anatomy permits. Neurologic surgeons are at relatively little variance concerning the general method of treatment. If pain is limited to some distribution of the trigeminal nerve, one may transect the root of that nerve by the subtemporal or occipital route. Sjöqvist,[5] in 1938, proposed a method of dividing the descending tract of the fifth nerve in the medulla. It will be recalled that as the sensory fibers of the fifth nerve enter the pons, a part of them course upward as the upper root and a large proportion course downward in the descending or bulbospinal tract. The tract extends as far downward as the second cervical segment. The fibers in this tract are axons of primary sensory neurones and synapse with secondary neurones in "the nucleus of the descending tract." The descending tract, therefore, is comparable to Lissauer's tract in the spinal cord and the nucleus of the tract is comparable to the substantia gelatinosa in the spinal cord. The fibers in the descending tract appear to be concerned with pain and temperature only. By inference the fibers in the upper root evidently transmit touch. Hence, while a section of the descending tract constitutes a section of primary neurones just as does section of the fifth root, the former has the decided advantage of eliminating pain with preservation of touch. Sjöqvist proposed that the section be made in the medulla at the level of the lowermost rootlet of the vagus nerve. The chief disadvantages of the procedure are injury to the restiform body and the fact that such sections always entail a certain amount of guesswork. The latter may be controlled by skill and practice and should not defeat a method which has merit. The former has been obviated in sufficient measure by making the section 10 to 12 mm. lower than the locus proposed by Sjöqvist. This modification in Sjöqvist's operation has been proposed and practiced by Grant and Weinberger[6] (1941) with no significant difference in the extent of resulting analgesia. In 1943, Grant[7] reported the results of thirteen medullary trac-

totomies by this method for the relief of cancer pain in the face. He recommended the method as a more desirable alternative than fifth root section for cancer pain not only in relation to its selectivity for pain and preservation of corneal integrity* but because in his experience there has been almost uniform absence of postneurectomy paraesthesia. (In the surgical treatment of tic douloureux, Grant's results have not been such as to warrant adoption of the modified Sjöqvist operation.)

The failure of the Sjöqvist operation to relieve the painful paroxysms of tic douloureux at first appears inconsistent in relation to the facial analgesia obtained. The failure, however, has been common experience. I believe that it may be explained by the fact that the intraoral zone is not made totally analgesic. I have given evidence[8-10] that demonstrates a strict relationship between the trigger zones (predominantly oral and immediately perioral) and the painful seizures. When the oral zone is made analgesic, the tic pain is abolished regardless of the referred distribution of the latter and if the trigger zone is not made analgesic the tic pain will not be cured. It may be recognized that the following statements have a general conformity with this proposition:

(1) As one severs the fibers of the fifth root from below in an upward direction (mandibular toward ophthalmic topography), anesthesia begins intraoral and progresses in a manner suggesting a segmental distribution but combined with some characteristics of peripheral branch distribution.[8] Hence, a partial section including the so-called maxillary-mandibular fibers by rendering the buccal zone analgesic (anesthetic?) abolishes the tic pain.[9,10] This is, no doubt, why Dandy "opined," as Sjöqvist put it[5] that the pain fibers in the fifth root near the pons were congregated near the operator. Obviously, such could not be the case or pain sensibility would be differentially lost in the entire distribution of the fifth nerve after partial section. It is the oral or "trigger zone" fibers that are nearest the operator. In personal

* In Sjöqvist's patients, corneal sensibility was greatly diminished or lost. This would seem logical because the medullary section eliminates pain sensibility most thoroughly on the ophthalmic side of fifth distribution and corneal sensibility is understood to be exclusively pain.

conversation Dr. Dandy said he meant these fibers in question to be "the pain fibers of tic douloureux." Dr. Dandy had characteristically made an accurate and significant observation leading to a correlation between partial section and tic pain, but I do not believe he had yet recognized the probable implication of the trigger bearing zone. Sjöqvist concludes from studies on degeneration of the tract after dividing root fibers that, "The portion of the root which is cut at the "Dandy operation" corresponds to the dorsal (or superior) part of the tract and nucleus. This portion consequently must be formed by the maximillo-mandibular fibers."

(2) As one destroys the descending spinal tract of the fifth nerve in a caudal to a cephalic direction, analgesia progresses in a strictly segmental fashion toward the buccal zone and the intrabuccal zone is the last segment to be affected.[11,10] It is, therefore, represented by the highest portion of the descending tract of the fifth nerve. This is unfortunate because medullary section of the tract cannot feasibly reach high enough to definitely include the intrabuccal zone. In short, medullary section severs all fibers of the fifth nerve except the appropriate ones in relation to "tic."

(3) The results that Sjöqvist[5] demonstrates in his patients bear out these facts. He did not often mention the effects on intrabuccal sensation. In the one patient (Case iv) in which he did mention it, however, there was "no impairment of the tongue." This was a case of most complete analgesia elsewhere, the section being at maximal height (midolivary). Moreover, corneal sensation was always markedly diminished or lost. This is not only an unfortunate result of the operation but is in keeping with the fact that the section eliminates thoroughly the ophthalmic zone.

Partial section of the fifth root by the subtemporal approach is, at present, the optimum operation for tic douloureux. In approaching the root extradurally, (the Frazier operation) a transient facial palsy has occurred in one of three to five patients. In two patients it has been total and permanent. It has made its appearance as long as five days after operation. It is somewhat of a mystery but it is attributed to injury of the seventh nerve through avulsion of the greater superficial petrosal nerve when the dura is stripped. By approaching the root intradurally and exposing it

through a window in the overlying dura the "bug-bear" of facial palsy has been totally obviated. The operation is actually less difficult and the approach almost bloodless. The results now leave very little to be desired.

In the case of pain in the face and throat, the ninth cranial root should be cut in addition to that of the fifth, or the alternative tract section just described. The fact that a ninth root section and a modified Sjöqvist operation can be done through a relatively small exposure in the posterior fossa and without great technical difficulty constitutes a distinct advantage. Moreover, by extending the central limb of a unilateral occipital incision (or the central limb of a cross bow incision) the posterior roots of the second, third and fourth cervical nerves may be severed for pain in their distribution.

Pain in the shoulder and upper extremity is in the large majority of cases an outcome of inoperable malignancy of the breast. Metastases to regional nodes, displacing and encasing the cords of the brachial plexus with eventual swelling, induration and lympho-edema of the shoulder and upper extremity associated with tenderness and severe pain is a pitiable situation. The plea for relief is an inescapable challenge to the neurologic surgeon. This territory is still unfortunately a sort of "no man's land." Neurosurgery has not yet been sufficiently adapted to the treatment of this zone and the surgeon is less confident than he is in respect to the other two corporeal territories. Section of the brachial plexus or intraspinal section of the sensory roots of the brachial plexus, even if such should eliminate the pain, presents the spectre of a dead and paralyzed extremity. In my experience, most of the patients whose mental and physical states were otherwise intact had great difficulty in resigning themselves to this ever present objective evidence of mutilation and the result fell short of being commendable. To make an issue of cutting only the sensory roots and preserving the motor is little short of quibbling. The fact that man can move a deafferented extrem-

ity on request, has in my experience provided little compensation and no consolation to the patient. There is something horrifying and gruesome about a totally dead upper extremity that dangles as an ever present senseless appendage. This reaction is far more intense in respect to the upper than to the lower extremity. It is granted that when there is no alternative, such a result is less an evil than a painful extremity which is just as useless. However, the rationalizing of those who do not have to swallow the medicine makes it taste no sweeter for those who do. Since our premise is to eliminate pain with minimum neurologic mutilation and melancholic despair, we are moved to search for a better alternative than deafferentation.

I have implied that even after section of brachial plexus roots or cords, pain in the extremity may not be abolished subjectively. I have encountered this paradox a number of times not only in respect to a deafferented upper extremity but in respect to other anesthetic zones. For example, M. S., a white woman of middle age, was admitted to the University Hospital, Iowa City, in December, 1941. She complained of severe pain in the right upper extremity as a result of inoperable carcinoma of the breast. The posterior roots from C-3 to D-1 inclusive on the right were cut. The extremity was totally anesthetic to objective tests but the patient continued to refer pain to it. Stroking the upper right chest with ones finger tips or certain places on the scalp or even stroking the sole of the right foot caused "tingling" sensations referred to the hand of the deafferented extremity. Because of pain in the axilla and upper right chest and the phantom pain in the extremity, anterior chordotomy was performed at C-8 on the left. After this the pain in the extremity largely disappeared but the phantom tingling persisted.

The first solution to this problem of the shoulder and upper extremity that would naturally suggest itself is section of the spinothalamic tract at a high cervical level.

My experience with this procedure has been disappointing on two counts. First, the level of analgesia has not reached high enough to satisfactorily relieve pain in the shoulder and upper extremity and second, the postoperative course has been stormy and beclouded with confusion, anxiety and respiratory embarrassment.

In relation to the level of analgesia, the author came to the following reasoning. After an experience with many anterior chordotomies at the level of D-1 (the sections being made between the eighth cervical and first dorsal roots) he concluded that the level of effective analgesia could never be brought higher than the skin level of D-5 by this method. Often the level of effective and total analgesia reached only to D-6. Hence, there was always an overlap zone of four to six segments between the level of cord section and the skin level of effective analgesia. Assuming that this same discrepancy in levels would apply to the cervical cord, it is clear why a section at C-2 or C-3 would fail to relieve brachial plexus pain. If a complete section of the spinothalamic tract were made at C-1, above the first cervical root, one might hope that at best the upper level of analgesia would reach the skin level of C-5 or C-6. Such a section in the cervical cord, however, is fraught with too great danger to be recommended. Moreover, division of the spinothalamic tract even in the medulla as proposed by Schwartz and O'Leary[12] has fallen short of establishing a high enough level of analgesia to solve this problem. Adams and Munro[13] tried the operation on three patients (two unilateral and one bilateral) and correlated their results with histologic studies made at postmortem. They concluded that although the operation was not especially dangerous in skilled hands, it did not result in analgesia in the neck, shoulders or upper extremities. They believed, therefore, that the method was not superior to high cervical chordotomy *plus section of cervical roots*. Their medullary sections were made at the inferior pole of the olive or at the uppermost

radicle of the eleventh cranial nerve. The resulting level of analgesia did not reach above the skin level of D-2.

It was in relation to this particular problem that I became interested in that uniform discrepancy between the level of section and level of skin analgesia. Commensurate with the existing knowledge of anatomy, I could only attribute this overlap to the proposition that pain fibers progress cephalad in Lissauer's tract by four to six segments before their secondary neurones cross to take their place in the spinothalamic tract. It was, therefore, assumed that if one sectioned the spinothalamic tract by anterior chordotomy at D-1 and also sectioned the fibers in Lissauer's tract at the same level but on the opposite side of the cord, he should be rewarded by a level of analgesia up to the skin level of D-1.* The results of this procedure adequately confirmed the hypothesis and have been presented in a preliminary report.[14] Two attempts to apply this principle to a cervical chordotomy at C-3 met with discouraging results in that the presumed section of Lissauer's tract did not raise the level of analgesia to include the distribution of the brachial plexus. The opportunity for further work along this line has not presented itself.

After high cervical chordotomy respiratory embarrassment has appeared to be attributable to interference with function of the phrenic nerve and even more. There has been impairment of diaphragmatic breathing and use of the accessory muscles of respiration. Since the impairment has appeared more serious than that after phrenic exeresis, I believe that high cervical chordotomy may interfere with central integration of respiration. The author performed a unilateral cervical chordotomy at C-2 on a young woman suffering from intractable pain in the chest on one side, the pain being due to inoperable sarcoma.

She located the pain as high as the supraclavicular fossa. The operation progressed quite smoothly and without complications in all respects but it was necessary to keep the patient in a Grinker respirator for the three months that she lived. Thus, aside from the hazardous nature of high cervical chordotomy, it fails to eliminate pain in the shoulder and upper extremity and hence the author believes that it has relatively little purpose. If there is indication to bring the level of effective analgesia as high as D-1 or D-2, he believes that a section in the medulla as described by Schwartz and O'Leary[12] and by Adams and Monro[13] is the procedure of choice by a wide margin. To eliminate pain in the shoulder and upper extremity, he believes that mesencephalic tractotomy such as that described by Walker[15] is a procedure which is thoroughly feasible and should be cultivated. He does not believe that high cervical chordotomy can be more than a chance taking operation to be followed by disappointing results. Few neurologic surgeons to the writer's knowledge have recommended this operation as having any value. Stookey[16] endorses it with surprising avidity but unfortunately does not discuss the level of analgesia attained nor does he cite a case that would support his claims. In the discussion of Stookey's paper, Penfield cited a patient on whom he did a unilateral anterior chordotomy at C-2 under local anesthesia. The patient died suddenly while the wound was being closed. Dr. Stookey replied that he had seen this before with bilateral chordotomies. To support the claims made for high cervical chordotomy, Stookey[17] refers to his own report of two cases of carcinoma of the breast in 1931. In one patient the chordotomy was unilateral at the second cervical segment and in the other it was bilateral at the second and third cervical segments. The report is short and the level of analgesia is not given. He refers also to the paper of Peet, Kahn and Allen.[18] These authors report the results of a bilateral cervical chordotomy on a young man with chronic infectious arthritis. The

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* Testing skin analgesia for accuracy of level should be carried out laterally or on the back because the supraclavicular nerves from the cervical plexus maintain algesia over the upper anterior chest.

operation was as follows: *On the right*, the fourth and fifth posterior cervical roots were cut and a section in the cord made at C-8. *On the left*, the fourth posterior cervical root was cut and a section in the cord made at C-3. The cord sections were made 4 to 5 mm. deep and extended from the base of the dentate ligament only to the point of emergence of the anterior rootlets. In their photograph of the patient, a line is drawn just above the clavicle on the right and at about D-2 on the left. It is stated that pain and temperature sense was lost below these levels. This is a most unusual result, especially in view of the limited breadth of the chordotomy. Moreover, subjective relief and interpretations in a patient with chronic arthritis are not comparable to those in a patient with carcinoma pain. To abolish intractable brachial plexus pain it is necessary that the distribution be definitely within the field of analgesia. My purpose in this critical review is to express a strong conviction that high cervical chordotomy should not be misrepresented as a procedure lacking in serious hazards or as a procedure having any specific or special value. I have been unable to find an incontrovertible report of a case in which the level of analgesia definitely included the distribution of the brachial plexus after a high cervical chordotomy alone, nor have I been able to attain such a result myself. Foerster and others[18] have been lenient toward unilateral high cervical chordotomy while strongly opposing the bilateral operation because of a prohibitive mortality. In deference to these disadvantages, it would seem timely and appropriate to step above the high cervical danger zone and cut the tract at a medullary or mesencephalic level.

Spinothalamic Tractotomy, Spinal. This procedure has been variously referred to as section of the spinothalamic tract, anterior chordotomy, anterolateral chordotomy and plain chordotomy. As with many other procedures in medicine and surgery, a haphazard name has become so indelibly identified with this operation, that it is not likely that more descriptive and specific nomenclature can easily take its place. When spinothalamic tractotomy was first proposed and carried out by Spiller and Martin,[19] it was not anticipated that a field of surgery involving sections in the spinal cord, medulla and mesencephalon would so develop that even "anterior chordotomy" would become a too broad and inclusive term. Section of the descending spinal tract of the fifth cranial nerve, Sjöqvist's operation, has been referred to as "medullary tractotomy" or "intramedullary tractotomy." This term might be applicable to and easily confused with section of the spinothalamic tract at a medullary level. Hence, in view of the need for some standard nomenclature, I would like to suggest the following: That section of the spinothalamic tract be referred to as *spinothalamic tractotomy* to be followed or perhaps preceded by a term designating the location of the section, i.e., spinal, medullary or mesencephalic; that section of the descending spinal tract of the fifth cranial nerve be referred to as Sjöqvist's operation or as *trigeminal tractotomy*. Reference in this way to the descending tract as *the* trigeminal tract seems permissible.

Section of the spinothalamic tract on a spinal level has evolved to take its rightful place among standard recognized operations of definite indication and worth. It was necessary that considerable work be done, both experimental and clinical, in order that expectations and limitations, disadvantages and sequelae, optimum methods and location of performance could be allocated to the operative procedure.

It is not necessary to give a detailed account here of the results and the knowledge accrued from the work that has been done. The results of my own experience and researches are given in previous publications along with references to the works of others.[20-25] It will suffice to express my conviction that the D-1 cord segment (between the roots of C-8 and D-1) is the over-all optimum location for *spinal-spinothalamic tractotomy*, largely because the highest

spinal level of analgesia is obtained and the operator is more assured of accomplishing his objective. Evidence has been deduced[20] to show that the tract mediating pain and temperature resides more anteriorly in the cord than Spiller supposed, at least at the D-1 level. Hence, in order to obtain a complete and high level of analgesia the section should be carried well beyond the anterior rootlet line. The writer believes that inaccurate topographical localization of the spinothalamic tract was responsible for an era of misconception in respect to the physiology of the tract and the value of tractotomy.

Many intriguing questions have been aroused concerning function of the spinothalamic tract and experience with many cases of chordotomy has made it possible to throw a little light on its nature. Some of the conclusions are briefly as follows: (1) The sensibilities of pain and temperature have always been impaired together and remained undissociable in all the patients studied; (2) the spinothalamic tract appears to be the only tract that mediates the sensation of pain; (3) the author has encountered no findings that would support a hypothesis of bilateral representation; (4) there has been no evidence that sensibility to pain could regenerate or recur in the distribution of the severed fibers of the tract; (5) the fact that a knife may be passed through the tract in the conscious patient without pain is a fascinating revelation which at present adds another mystery to nervous integration; (6) at present it is reasonable to postulate that section of the spinothalamic tracts alone would result only in a loss of the sensibilities to pain and temperature, this being their specific function and *raison d'être*. The patient is aware of the deficiency only through appropriate stimulation and (7) the sensation of "itch" is undoubtedly a complex modality of pain and is completely abolished on section of the tract.

The untoward sequelae associated with spinothalamic tractotomy result from injuries to neighboring tracts which are, as yet, unavoidable. These complicating factors are minimal and often totally absent when the section is unilateral. They always appear to some degree when the section is bilateral. Those deserving prime consideration are: (1) Subjective and objective weakness of the lower extremities which is recoverable in a week to a month. One defect may remain indefinitely, a subjective weakness of the knees, probably due to impairment of the static, antigravity reflex integrated through the vestibulospinal tract;[23] (2) weakness of bowel and bladder sphincters requiring catheterization for one to two weeks; (3) loss of the possibility of orgasm in both male and female and (4) the writer has seen two patients who maintained a permanent and severe orthostatic hypotension after a bilateral operation.

As one might expect the debilitation accompanying cancer retards recovery and augments the disadvantages. The latter, however, by no means contraindicate the operation if pain is unbearable and otherwise intractable. While one should unequivocably always perform the unilateral operation if at all reasonable, it is equally as wise to do the bilateral operation if the migration of pain to the opposite side appears inevitable. Multiple stage operations in the management of a patient with intractable cancer pain soon makes the treatment as bad as the disease.

I do not believe that statistics in the form of percentages have any significance here. It is sufficient to report that the operative mortality from spinal chordotomy has been less than 1 per cent. The tenure on life has ranged from two months to five years and prognoses in this respect are notoriously inaccurate. Almost without exception the pain has been completely abolished or satisfactorily relieved. In the bilateral chordotomies some degree of motor weakness in the lower extremities and at least a transiently atonic bladder have resulted in every patient. The degree with which these deficits have occurred

correlates roughly with the debilitating effects of the cancer. It would appear to me that the comfort which the patient has during his limited time is the more significant objective and measure of success rather than the number of days or months of survival. It should be kept in mind that this discussion involves only the intractable pain due to cancer. The long term objectives and methods of treatment of pain from other causes are considerably different. The unwonted neurologic deficits are less prominent and when they appear after chordotomy, recovery from them is more rapid and substantial.

PREFRONTAL LEUCOTOMY

Although I have no personal experience in prefrontal leucotomy as a method of relieving intractable pain, I believe that its merits quite possibly may supersede those of the methods that have been described. Judging from the knowledge and experience that has and is being accrued from this operation and staking an abstract view anent the influence of prefrontal integration on the "intractability" of pain, it appears to be rational and promising. Prefrontal leucotomy looms as the third stage in the evolution of surgical methods in the treatment of pain. In the first stage, focus was upon the most immediate expediency and thus amputation and peripheral nerve section prevailed. In the second, an attempt is made, (i.e., by chordotomy) to interrupt specifically the pain-conducting system of fibers with otherwise minimal neurologic deficit. This leaves much to be desired, however, and may quite possibly prove to be transitional to a third stage in which the objective is to treat not only the unpleasant sensation of pain but also the psychic reaction to it and its sinister cause. The proposal is intriguing and provokes an endless chain of philosophic debates. Whether the wiggling of an injured earthworm is an expression of pain is an age-old controversy.[26] Whether decorticate man could suffer, will by nature of the problem always remain an abstraction.* But it is most reasonable to prophesy that if pain (in a broad sense with mental anguish and agony) can be reduced to a subconscious or unconscious reflex or projection, surgery will have reached the acme of achievement. This in a measure is the design of prefrontal leucotomy and the reports are encouraging.[28]

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* Walker[27] touches upon this abstract problem with some refreshing notations and a cautious span between physiology and philosophy.

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SURGICAL TREATMENT OF EPILEPSY*

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MANY different pathologic conditions may be associated with convulsive seizures, so that an accurate diagnosis must be made in every case before any type of therapy, medical or surgical, can be rationally prescribed for the attacks. The etiologic factor may be suspected from the history but often cannot be demonstrated even after complete physical, neurologic, electroencephalographic, pneumoencephalographic and arteriographic examinations. In childhood, idiopathic epilepsy associated with a cerebral dysrhythmia is common but in adult life a convulsion is much more likely to be a symptom of a focal brain lesion.[63] Even if all the examinations mentioned fail to disclose a cause for the attack, the patient must still be suspected of harboring a small cerebral lesion which may later give more definite evidence of its presence.

With the exception of those cases of brain tumor, which require immediate surgery, the therapy of both idiopathic and symptomatic epilepsy is primarily medical. Only when anticonvulsive drugs in doses approaching the toxic levels have failed to control the attacks is surgical intervention justified.

IDIOPATHIC EPILEPSY

Trephination. Historical writers have produced evidence that the earliest operation for the relief of epilepsy was simple trephination. Numerous trephined skulls have been found in the ruins of ancient civilizations. This procedure was presumably done to release devils considered to be irritating the brain. Although the demonological conception has passed, the use of trephination in the treatment of epilepsy has not been abandoned entirely. Cutter[7] discusses five cases reported by Dudley in 1828 in which trephination and decompression was done. But in general, neurosurgeons concur with Penfield[37] who states that the results of subtemporal decompression do not justify the procedure in essential epilepsy.

Pneumoencephalography. Therapeutic effects of pneumoencephalography have been repeatedly reported since the introduction of this technic. In idiopathic epilepsy the procedure has not changed the course of the disease.[60] It is true that some patients have fewer attacks after the air injection but in most cases this improvement may be attributed to better medical management of the case. Thus Penfield[37] reports five of ninety-six patients to be seizure-free for an average of 2.8 years after pneumoencephalography. These patients were under sixteen years of age and had had attacks less than four years.

Freeing of Corticodural Adhesions. Ney[34] ascribes some cases of idiopathic epilepsy to local cortical tension due to traction by corticodural attachments at the vertex. Two hundred twenty-five patients with idiopathic epilepsy were operated upon and the adhesions relieved. The results were said to be good but the follow-up is not adequate to evaluate this procedure. Scarff[52] believes that there is a specific epileptic syndrome caused by anomalous pacchionian granulations extending into the motor area. Division of these granulations thus freeing the corticodural adhesions relieves many of the patients to some extent.

Miscellaneous Procedures. On the assumption that the sympathetic supply to the cerebral vessels is unstable in idiopathic epilepsy, sympathectomy has been performed. However, Penfield states, "Sym-

pathectomy, however complete, does not make seizures impossible and is hardly justifiable as a treatment for epilepsy except in the presence of unusual evidence of abnormality of the sympathetic nervous system."[37] Removal of the carotid body and denervation of the carotid sinus have also been used in the treatment of epilepsy but Penfield[37] reports that the procedure is of no value unless specific abnormal hyperactivity of the carotid sinus exists.

With the purpose of preventing the convulsive seizure becoming generalized and thus preventing loss of consciousness in epileptics having a unilateral beginning of their attack, Van Wagenen[37] divided the corpus callosum. Six of ten patients so treated have had no generalized seizures since operation. Van Wagenen[38] has also made a hypothalamic puncture at the upper end of the pituitary stalk to induce dehydration by diabetes insipidus. In one case no convulsions had occurred at the time of the report.

In psychomotor epilepsy, negative spike foci have been found at the tip of the right or left or both temporal lobes.[17] Ablation of the tip of the temporal lobe and the focus has been carried out but the results are not yet known.

SYMPTOMATIC EPILEPSY

Many disorders of the brain are associated with convulsive seizures. The most common conditions include such congenital abnormalities as cerebral dysplasias (aplasia of cortex, microgyria, etc.) and vascular anomalies, degenerative states, inflammatory diseases, traumatic alterations of the brain and neoplasia. But not all patients suffering from these conditions have seizures. Probably less than 40 per cent are so afflicted. What factors render certain individuals susceptible? The complete answer to this riddle is unknown but some epileptogenic components are apparent.

i. *Location of the Lesion.* Cerebral lesions about the Rolandic area are prone to be associated with convulsions while polar lesions rarely have epileptic concomitants.

In cerebral neoplasms and scars this factor has been studied in some detail.[2,15,19,20,29,35,41] List considers the histologic type of glioma to play an important rôle irrespective of the location of the tumor.[29]

ii. *Severity of Cortical Damage.* Factors which tend to cause extensive cortical damage have been correlated with a high incidence of epilepsy. Thus penetrating dural wounds, infected wounds and extensive wounds are associated with convulsions about twice as often as a general, unselected group of head injuries. Retained foreign bodies, however, appear to have no relationship to the development of convulsions.[2,4,14,19,30,47,48,59,61]

iii. *Heredity.* Is there an inherent predisposition to convulsions? The problem has been more adequately studied in post-traumatic epilepsy than other types of symptomatic epilepsy. Cobb gives statistics bearing on this question.[5] In 250 normal control subjects epilepsy occurred in 2.6 per thousand of 1,896 parents, siblings and offsprings. In 1,086 non-traumatic epileptics, epilepsy occurred in 21 per thousand of 9,139 relatives. In 235 patients with epilepsy who had suffered head trauma, there were 14 per thousand epileptic relatives. This figure is considerably higher than that occurring in the group of normal controls. The figures obtained in the group of post-traumatic epileptic patients studied at Cushing General Hospital differ from those of Cobb.[47] In this series only 4.5 per cent of the patient's relatives had convulsions compared to an incidence of 3.4 per cent in normal families and 17 per cent in epileptic families.[27] Ziskind[66] doubts that there is an inherited predisposition to epilepsy since it would have to be present in from 20 to 40 per cent of patients with head injuries and tumors. The epileptogenic factors are obviously not yet completely understood.

Type of Seizure in Symptomatic Epilepsy. There is nothing peculiar in the seizure of symptomatic epilepsy to distinguish it from that associated with idiopathic convulsions. In a series of cases, focal attacks are

much more common in symptomatic than in the idiopathic epilepsy.[2,14,47,61] The mode of onset of the seizure is usually an indication of the site of the lesion within the brain. Thus lesions in or near the motor area usually induce attacks beginning in the contralateral arm or hand, less commonly in the face and rarely in the leg. Sensory beginnings of the attack indicate the position of the lesion in the parietal, temporal or occipital lobes. The aura or onset of the attack is usually an excitatory phenomenon but paralytic manifestations are seen. Thus the most frequent aurae are twitching of the face, eyes, fingers, etc., but occasionally the onset is indicated by an inability to talk or a weakness or heaviness of the arm. Similarly in the sensory sphere, the aura may be excitatory or paralytic. For example, visual aurae may consist of a blurring of vision in one or both visual fields. This is frequently described as a black ball, dots, circular curtain moving across the visual field in an undulating or pulsating fashion. Frequently this scotoma begins in the periphery of a hemianoptic sector, proceeds across the vertical meridian to involve the normal field causing a complete loss of vision for a few minutes. With the onset of blindness a generalized attack sometimes develops. The visual aura may also consist of white or colored "lights" in the affected visual fields. When lateralized the hallucination occurs in the visual field contralateral to the cerebral wound.

Vertiginous aura manifested by dizziness just before a generalized attack is not an uncommon complaint and in some cases a true vertigo is present. Auditory aura and epigastric aura may occur at times. The epigastric aura of a feeling of uneasiness in the pit of the stomach, nausea, a choking sensation, cramping abdominal discomfort and vomiting are not so frequently encountered as in idiopathic epilepsy. Complex and bizarre aura may also occur. These may consist of combinations of auditory, visual and motor aura.

These focal manifestations may constitute the entire attack or they may progress to a generalized convulsive seizure. A true Jacksonian seizure is not often seen in symptomatic epilepsy nor are petit mal attacks as common as in the idiopathic seizures.[14,47,54,55,61]

ELECTROENCEPHALOGRAPHIC FINDINGS IN SYMPTOMATIC EPILEPSY

The electroencephalogram usually distinguishes the symptomatic from idiopathic epilepsies. It is based upon evidence of focal brain damage such as slow delta waves with superimposed sharp, spiky waves. This question has been most extensively studied in the post-traumatic epilepsies which will be discussed in some detail.

In a study of eighty-one cases of post-traumatic epilepsy Jasper and Penfield[23] found that in 90 per cent the electroencephalogram revealed a local area in one hemisphere from which random spikes or sharp waves were most prominent, usually upon a background of random delta waves. Slow waves alone, they believed, were not a reliable guide and positive electroencephalographic evidence of a focal lesion was present only when spikes or sharp waves were repeatedly recorded from the same discrete area.

In an early report Williams states that the character of the changes seen in the electroencephalographic record in traumatic epilepsy appear to be the same as in idiopathic epilepsy.[64] In a later report, however, he states that focal abnormality is found in a large proportion of cases of traumatic epilepsy in contrast to the group of idiopathic epileptics.[65] An abnormal electroencephalogram persisting after a head injury does not necessarily increase the likelihood of traumatic epilepsy; but if paroxysmal outbursts are present, the likelihood is increased by four times. If a patient's electroencephalogram shows larval epileptic outbursts, epilepsy is almost certain to supervene. Gibbs, Wegner and Gibbs[16] found that focal electroencephalographic abnormality is four times as

common in post-traumatic epileptics as in unselected epileptics and that the focal electroencephalographic abnormalities correlate with focal seizures. Focal seizure—discharges are twenty-one times as common among post-traumatic epileptics as among head injury patients without seizures. Roseman and Woodhall[49] reported paroxysmal electroencephalographic changes in fifteen cases of posttraumatic epilepsy; in twelve cases with focal seizures localized electroencephalographic discharges were present. In four of the fifteen patients with convulsions abnormal electroencephalographic foci were recognized first. However, there were another fifteen cases in the series with similar paroxysmal abnormal electroencephalographic changes but without clinical convulsions.

Kaufman and Walker[24] studied the electroencephalograms of 241 cases of post-traumatic epilepsy and of eighty-three cases of severe head injury but without seizures. The records were abnormal in 91.3 per cent of the epileptic and 77.1 per cent of the non-epileptic group. A focal abnormality was present in 83.8 per cent of the epileptic cases and in 67.5 per cent of the control group. The most common focal abnormality in both groups was the presence of slow waves, but this was much more frequent in the epileptic (73 per cent) than in the non-epileptic group (48.2 per cent). Paroxysmal abnormalities consisting of sharp waves or spikes which are usually considered indicative of epilepsy, occurred about equally (approximately 20 per cent) in the epileptic and control series. (Fig. 1.)

The epileptic character of a focus may be brought out by metrazol activation[25] or in sleep records.[18] Following a rapid intravenous injection of 200 mg. (2 cc.) of metrazol, within thirty to sixty seconds focal epileptic abnormalities usually appear in the electroencephalogram. This focal discharge consists of slow waves or humps having a frequency less than the alpha rhythm or single or multiple spikes, at times occurring rhythmically, giving the pattern of a localized electroencephalographic seizure. (Figs. 2 and 3.) This type of focal discharge occurs in about 67 per cent of a series of cases of post-traumatic epilepsy. If the procedure is repeated on another occasion, approximately one-half of the cases having no change on the first examination will have electroencephalographic alterations on the second trial. Abnormalities present before activation are usually aggravated by the metrazol. In about 88 per cent of the positive cases the focal electroencephalographic alterations induced by metrazol are paroxysmal. In 10 per cent of cases generalized electroencephalographic alterations consisting of single or multiple slow waves or spikes are present simultaneously in tracings made from several parts of both sides of the head, giving a pattern of a generalized electroencephalographic seizure.

If the metrazol activation is made after the patient has had no anticonvulsant medication for several days, clinical seizures similar to the patient's usual attack will occur in about 15 per cent of the cases. Clinical seizures have not occurred when anticonvulsant medication was not interrupted, but in a small percentage of cases the patient may experience a sensory or motor aura.

The epileptic manifestations may be brought out by spontaneous or induced sleep while the patient is having an electroencephalographic record made.[18]

EPILEPSY ASSOCIATED WITH BRAIN TUMOR

Convulsive attacks are a common symptom of brain tumor. Sargent[51] reported an incidence of convulsions of 30.9 per cent in a series of 270 patients. Parker[35] in a series of 313 patients with cerebral tumor found that sixty-seven (21.4 per cent) had major epileptic convulsions. Dowman and Smith[10] found that seizures occurred in 39 per cent of 100 cases of intracranial growth. In a statistical survey of Bailey's cases from the University of Chicago

4 - 1

1 - 3

3 - 5

5 - 7

7 - 8

2 - 9

9 - 7

7 - 10
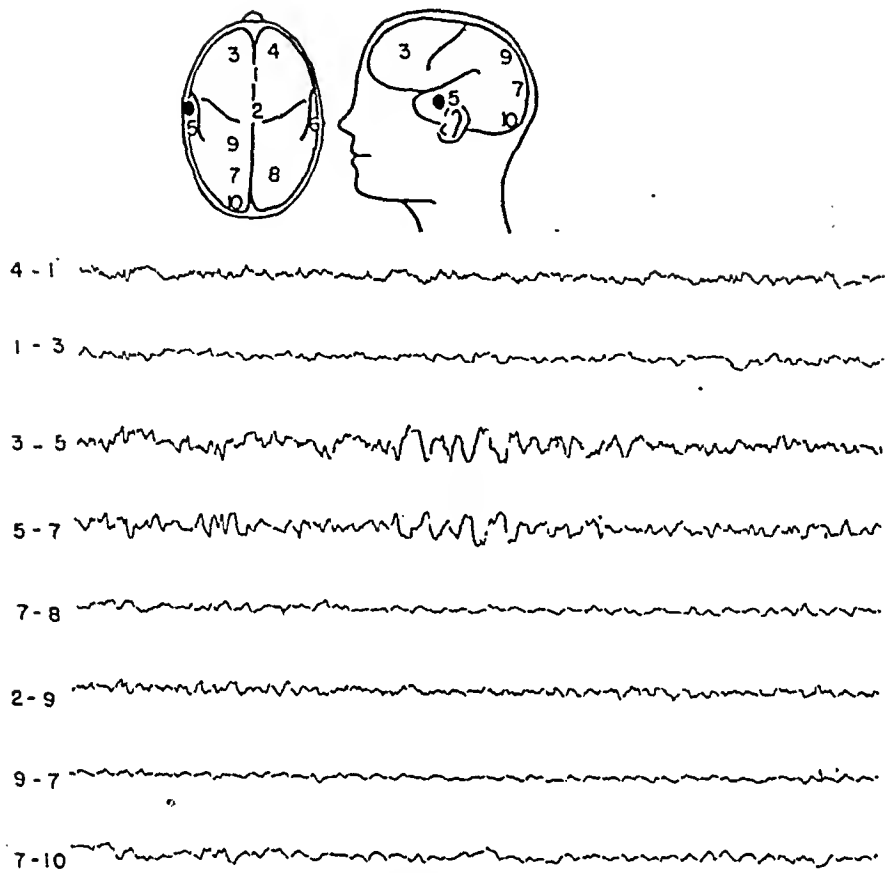
FIG. 1. Electroencephalogram of a patient suffering from post-traumatic epilepsy. The slow wave focus at the site of the small cranial wound just above the ear is evident. The leads are indicated on the traces. The horizontal line at the base represents an interval of one second and the vertical line a calibration of microvolts.

METRAZOL ACTIVATION
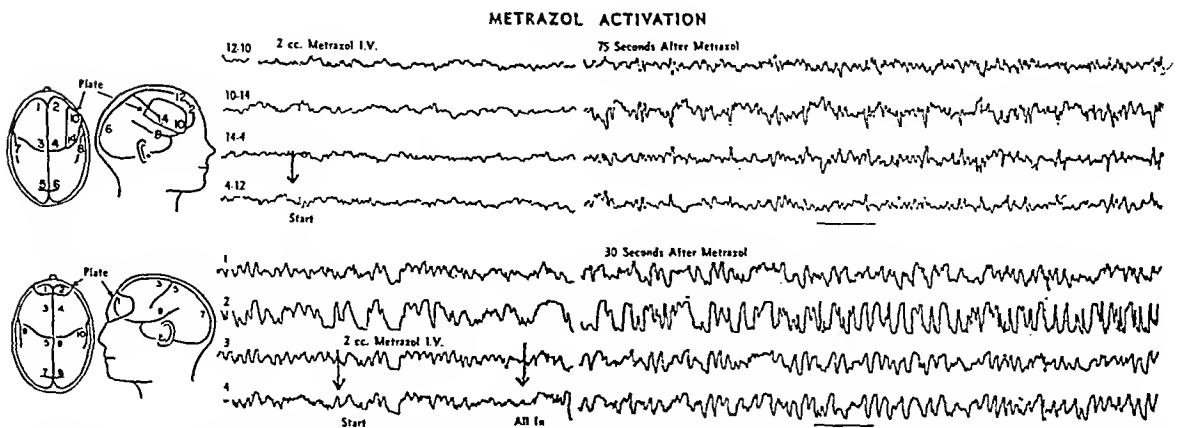
FIG. 2. Records showing the effect of metrazol activation in post-traumatic epilepsy. In the upper record the spiking at point 14 is apparent. In the lower record the marked slowing of the cortical rhythm with particular irregularities in the second trace is evident. An interval of one second is indicated by the horizontal line at the base.

Ley and Walker[28] found that 25.7 per cent of the patients suffering from tumor of the brain had generalized convulsions. Penfield et al.[41] report an incidence of 51 per cent in intracerebral and 62 per cent in extracerebral neoplasms.

the greater is the likelihood of convulsive seizures developing.[29,41] Gibbs[15] and Groff[20] have found that a tumor of the temporal, parietal or frontal lobe produces seizures more frequently than one in another region. Infratentorial and pituitary tumors
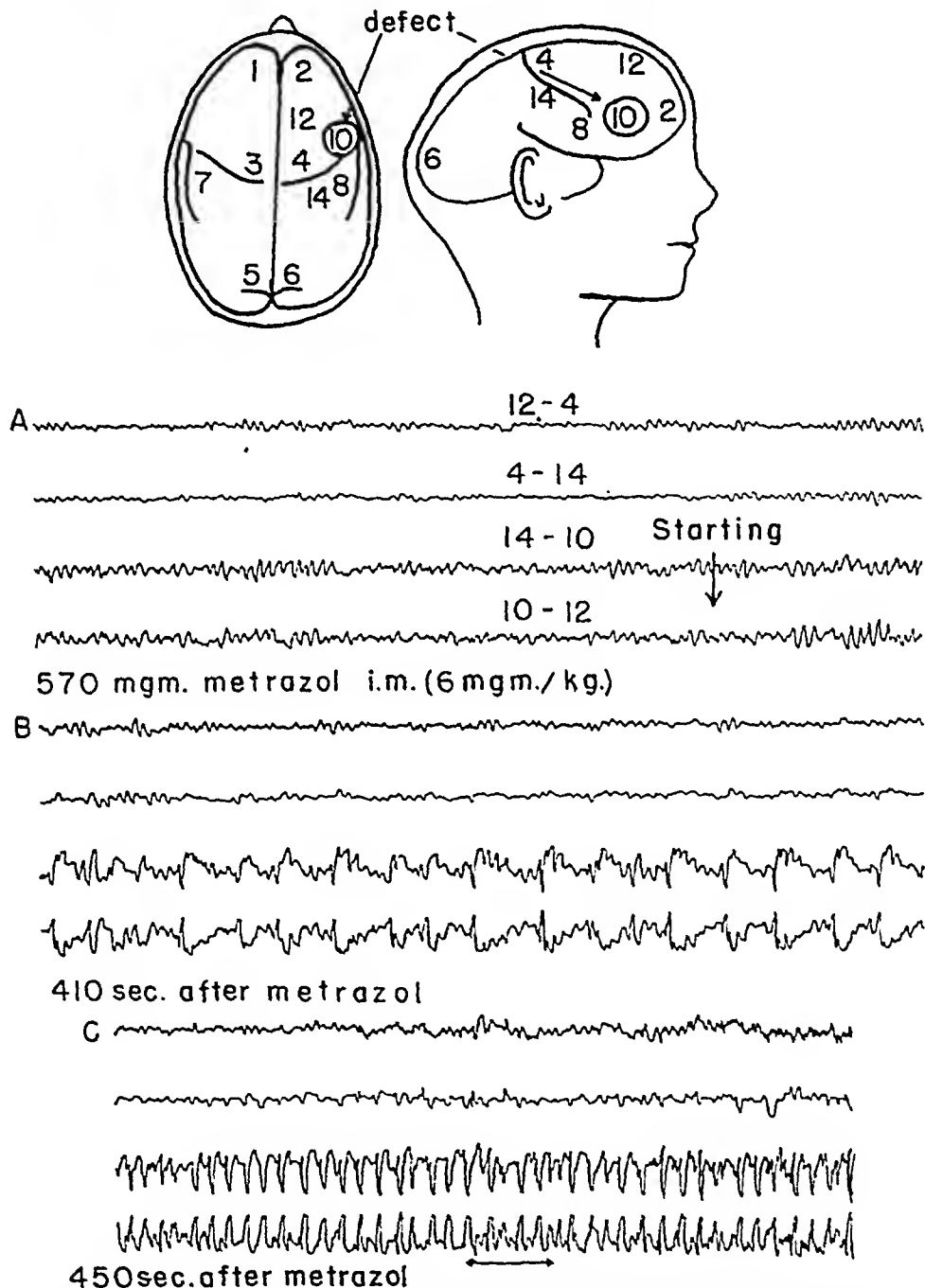
Fig. 3. Electroencephalogram to show the activating effect of intramuscular metrazol. The leads are as indicated. The spiking from point 10 is well shown in the lower two sets of tracings. An interval of one second is indicated by the horizontal line at the base.

The incidence of convulsive attacks in patients with cerebral tumor varies with the type and location of the neoplasm. The nearer the tumor is to the central sulcus

rarely are associated with convulsions.[41] Tumors with a long life history are likely to be associated with convulsive seizures. In Penfield's series of cases[41] 37 per cent

FIG. 4. Angioma of left inferior frontal region inducing convulsive seizures.

of the glioblastomas had seizures, 70 per cent of the astrocytomas, 68 per cent of the meningeal tumors and 92 per cent of the oligodendrogliomas. List found a similar relationship between epilepsy and histologic type of tumor.[29]

Removal of the tumor frequently abolishes the attacks. This result may be enhanced by care at the time of craniotomy that no devitalized or macerated cerebral cortex is left at the site of the tumor. Penfield et al.[41] report that of twenty-nine epileptic patients with meningiomas, nineteen continued to have seizures after removal of the tumor. In a series of twenty-one non-epileptic patients with meningiomas nine had developed seizures after operation. In a group of twenty-seven epileptic patients with astrocytoma, twenty-three continued to have seizures; of six patients without preoperative seizures, three later developed seizures.

Following operation, a patient who has had convulsive attacks preoperatively should continue to take anticonvulsant medication. If he remains attack-free for a period of two to three years, the anticonvulsant medication can then be gradually discontinued. The question arises as to whether all patients with brain tumor should receive postoperative anticonvulsant medication since a certain percentage of them will develop seizures. This is a matter of individual choice but in most cases medication is not given until the patient has developed attacks.

EPILEPSY ASSOCIATED WITH CONGENITAL LESIONS

Congenital lesions of the cerebrum are numerous in type and degree. Epilepsy is an infrequent symptom of certain of the congenital lesions although in the localized aplasias and especially the vascular abnormalities it is particularly common. (Fig 4.) Usually included in the congenital abnormalities are cerebral lesions incident to delivery such as cerebral hemorrhage, contusion or laceration and vascular occlusion. These injuries cause the formation of atrophic gyri and cysts.

The treatment of epileptic seizures secondary to congenital lesions is primarily medical. With the use of appropriate doses of anticonvulsant medication many of the cases can be controlled. Roentgen therapy is of value in some cases of vascular abnormality. The indications for surgery in the treatment of epileptogenic congenital lesions are few even in many of the cases not controlled by medication. Frequently the damage to the cerebrum is diffuse and widespread so that little can be accomplished by surgical intervention. Surgical therapy is indicated in those cases not controlled by adequate anticonvulsant medication in which there is neurologic, roentgenologic or electroencephalographic evidence of a focal lesion. The congenital vascular abnormalities constitute a special problem. The former high mortality involved in their surgical treatment has been reduced by present methods of hemostasis and adequate blood replacement.[46]

EPILEPSY ASSOCIATED WITH INFLAMMATORY DISEASES

There are a number of types of inflammatory disease of the central nervous system which may result in convulsive seizures. The most common of these are the pyogenic infections. This includes the acute pyogenic meningitides, chronic arach-

noiditis secondary to a meningitis and pyogenic brain abscess. Seizures occurring in an acute meningitis are, like fever and delirium, only temporary symptoms of the disease. Only rarely will a chronic convulsive state develop secondary to pyogenic meningitic involvement. Seizures are common symptoms of an acute brain abscess and not infrequently continue when the abscess has been aspirated, drained or ablated. The convulsions may begin a number of years after drainage of the abscess.[1,6,21,23] Occasionally a chronic encapsulated abscess which was never surgically treated may remain for years unrecognized with symptomatic convulsive seizures. (Fig. 5.)

The convulsive seizures of neurosyphilis are well known. Tuberculomas are frequently diagnosed as cerebral tumors and may be associated with convulsive seizures. The yeasts and fungi may invade the central nervous system producing a chronic meningitis with occasional epileptic attacks. An amebic abscess is sometimes found within the cerebrum. Certain parasitic infections such as echinococcus, cysticercus and the shistosomes involve the cerebrum with convulsive seizures as a common symptom. These infections, becoming more common in this country, may be diagnosed in some cases by calcification in the multiple lesions.[8,9]

The treatment of the epileptic seizures in these various inflammatory diseases consists of specific therapy directed against the causative organism and anticonvulsive therapy.

If medication does not control the seizures, a surgical exploration and ablation of the epileptogenic zone is indicated.

EPILEPSY ASSOCIATED WITH CEREBRAL SCARS

Following a cerebral wound, a firm scar forms binding together the cerebral cortex and dura mater. In many cases a layer of blood in the subdural space becomes organized forming a membrane of varying thickness. At the time of opera-

tion for post-traumatic epilepsy this subdural membrane is found firmly adherent to the dura mater. It may be 2 to 10 mm. in thickness and quite extensive overlying as much as one-half of a hemisphere in some cases. The brain adjoining the meningocerebral scar in some cases appears normal, but frequently it is yellow or brown, softened and cystic. The arachnoid is often thickened and opaque, occasionally containing white calcified plaques. Because of atrophic gyri, arteries and veins usually concealed in sulci may present on the surface of the brain.[61] (Figs. 6 and 7.)

Histologically a meningocerebral cicatrix consists of connective tissue with collagen fibers and blood vessels. The cicatrix may contain neuroglia cells of the piloid variety but usually no ganglion cells.[38,42] Penfield has described the anastomosis of the vascular channels in the connective tissue with the vaso-astral network of the brain. Because of this anastomosis, as the cicatrix contracts there is a resultant pull and traction on the brain.[36] Between the scar and normal gray matter is an intermediate zone of partially destroyed gray matter in which there is evidence of progressive neuronal destruction in small patches surrounding blood vessels. The presence of compound granular corpuscles and changes in the neuroglia are said to be evidence of the progressive nature of the lesion.[43] This histologic picture is the same whether the patient does or does not develop epilepsy.[3,26,43]

The distortion of the brain due to the cerebral scarring is well illustrated in pneumoencephalograms. The most common pneumoencephalographic finding in the cases of penetrating head injuries is a bilateral ventricular enlargement with an outpouching at the site of the skull defect. In a series of 109 patients with post-traumatic epilepsy who had pneumoencephalograms made from three months to three years after their head injury, this type of pneumoencephalographic appearance was present in sixty-three cases. In

FIG. 5. A calcified capsule of a chronic abscess secondary to otitis media. A, lateral roentgenogram of the skull; B, extirpated abscess.

twelve cases there was unilateral ventricular enlargement with an outpouching and in eight cases a simple unilateral ventricular dilatation on the side of the lesion.[61] (Fig. 8.) The subarachnoid space, usually on the side of the lesion, is poorly demonstrated in many cases, but occasionally cysts or dilated sulci are visualized. The pneumoencephalograms of patients suffering from closed head injuries were either normal or had symmetrical ventricular dilatation. Of the entire group of 109 patients, only fourteen pneumoencephalograms were reported as normal. These ventriculographic changes are not peculiar to cases of post-traumatic epilepsy. Practically the same findings were present in a non-selected group of head injuries re-

ported by Troland et al.[56] although the incidence of severe ventricular distortion was higher in the epileptic series than in the unselected one. Convulsive attacks may occur immediately after a head injury or they may make their appearance many years later. For convenience most authors divide post-traumatic epilepsy into immediate (attacks in the first twelve to twenty-four hours), delayed, (attacks in first four weeks) and late epilepsy. Penfield[44] believes that the "early fit" should not be included in the discussion of post-traumatic epilepsy. It is true that a large percentage of patients with immediate attacks do not continue to have recurring seizures. In Penfield's group of 407 cases of civilian head injury there were fourteen patients with early seizures out of which only 4 or 28.5 per cent developed traumatic epilepsy.[44] In Wagstaffe's 377 cases of gunshot wounds of the head five of twelve patients with early seizures became chronic epileptics.[59] Ascroft[2] states that in about one-third of the patients having fits, the attacks are more or less of a transient character. Maltby[30] reported thirty-four cases of convulsive dis-

FIG. 6. Large meningocerebral cicatrix causing traction of the Sylvian vessels toward the scar.

order following penetrating craniocerbral injury. In one-third of these cases the attacks occurred within a week or two of the injury; three of these patients continued to have seizures.

In the majority of cases of post-traumatic epilepsy the attacks begin within the first few years. In a series of 500 cases of gunshot wounds of the head, Gliddon[19] states that 43 per cent of the cases appeared in the first year following injury. The average latent interval in twenty-six cases of post-traumatic epilepsy reported
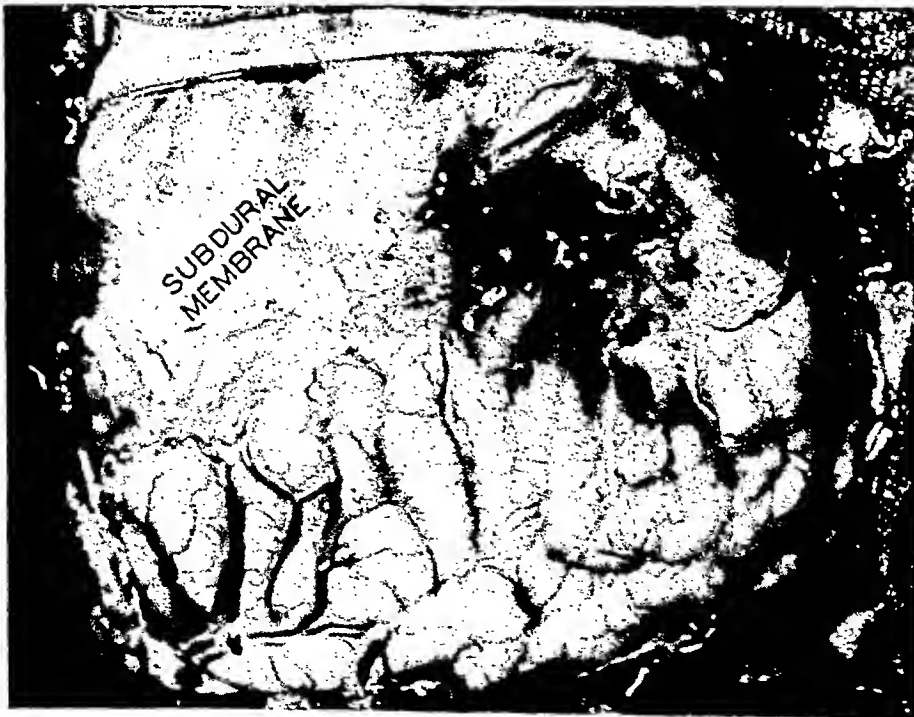
FIG. 7. Meningocerebral cicatrix with adjacent thin, subdural membrane.

Fig. 8. Lateral pneumoencephalogram to show the marked trigonal dilatation and out-pouching in the direction of a skull defect in a patient suffering from post-traumatic epilepsy.

by Symonds[55] was two years, nine months. In seventeen cases it was three to eighteen months and in nine cases two to sixteen years. In another group of cases reported by Elvidge[11,12] the average time of appearance of attacks of forty cases of grand mal was 5.3 years. Twelve appeared within the first year, twelve more in one to five years and ten more in five to ten years. One case occurred twenty-five to thirty years after injury. The average time in sixteen cases of petit mal was 5.82 years. Ascroft[2] states that at the end of the second year the rate of onset reaches a very low figure and that only about one patient in twenty-seven developed epilepsy between the fifth and twentieth years. In a series of 246 cases of post-traumatic epilepsy treated at Cushing General Hospital, 27 per cent of the cases had the first seizure within three months and 30.7 per cent between three and six months after injury.[47]

The reported incidence of post-traumatic epilepsy varies considerably. This is probably due to several factors. There are differences in the type of head injury occurring in the various groups of cases. Criteria vary as to what to include as a convulsive seizure. Some figures include immediate attacks while others omit these. In a series of 500 cases of gunshot wound of the head including all cases having had one incident labelled epilepsy at any time following the head injury, Gliddon reports forty-nine cases of epilepsy, an incidence of 9.8 per cent.[19] In a similar series of 377 cases with the time of onset varying from two months to seven and one-half years, Wagstaffe[59] gives the same incidence, 9.8 per cent. In other reports of series of cases of gunshot wounds of the head, the incidence of convulsive seizures is as follows: Rawling,[48] 452 cases, 22 per cent; Stevenson,[54] 17,300 cases, 1.5 per cent; Sargent,[51] 18,000 cases, 4.5 per cent; Cushing,[4] 34 cases, 32 per cent; Maltby,[30] 200 cases, 17 per cent. In a series of 407 cases of civilian head injury, Penfield[44] found an incidence of epilepsy of 2.7 per cent. From these figures it can be seen that the incidence of post-traumatic epilepsy including all types of head injury varies from 1.5 to 34 per cent.

SURGICAL TREATMENT OF SYMPTOMATIC
EPILEPSY

When medical management fails to
control convulsive seizures due to a cere-
bral lesion, surgical procedures may offer
relief if no contraindications exist. It is
not wise to subject individuals who are
aphasic, paraplegic or quadriplegic and
confined to bed, to a serious operation for
the relief of convulsive seizures which
play only a minor rôle in their disability.
If a patient is mentally disturbed or im-
paired to such a degree as to render him
or her incompetent of following medical
advice, the advisability of operation is
doubtful. Because a cortical resection con-
stitutes a major surgical procedure, the
general condition of the patient should be
as good as possible prior to operation.
If the primary wound was infected or did
not heal by primary intention, it is well
to delay cortical exploration for at least
six months to a year after the wound has
healed. When the operative procedure is
then done, chemotherapeutic agents, peni-
cillin and sulfadiazine should be used pre-
and postoperatively.

Just as in the cases of idiopathic epi-
lepsy, a number of surgical procedures
have been tried in the treatment of symp-
tomatic epilepsy. Meredith[32] has described
an operation for internal communicating
hydrocephalus following certain traumatic
or inflammatory lesions associated with
seizures. The operation consists of a
transcortical incision through a relatively
unimportant portion of the less dominant
cerebral hemisphere leaving a large stoma
into the lateral ventricle. At the same time
the choroid plexus is removed from the
opened ventricle. Siris[57] reported five cases
of focal epilepsy treated by encephalo-
myopexy. Three patients had not had
seizures for five to seven months post-
operatively and the other two had dimi-
nished frequency and intensity. Marsh[31]
stated improvement occurred in some
cases of traumatic epilepsy in a series
which had been given radiation therapy.
It is difficult to evaluate these different

procedures because of the small number
of cases reported.

The usual procedure used for the treat-
ment of post-traumatic epilepsy is the
surgical removal of the epileptogenic focus.
Until recently it was thought that the
lesion, scar, tumor, softening, etc., was
the focus responsible for the seizures, but
with the use of electrocorticography it has
now been clearly shown that the marginal
cortex is the source of epileptic discharges.
In a recent discussion of post-traumatic
epilepsy Penfield[45] stated: "We must
identify more definitely, not the gross
lesion but the actual focus of neuronal
hyperirritability. It is the focus in which
electrographic spikes and sharp waves
originate and which causes the attacks."

The question arises if the epileptogenic
focus is stable and if it does not with the
passage of time set up other epileptogenic
foci? These questions cannot be answered
with assurance at this time although the
available evidence favors the stability
and constancy of an epileptogenic focus.
Whether secondary foci will form if the
primary lesion is left, remains for further
research. Perhaps that is the explanation
for certain failures.

Cases suitable for this type of surgical
procedure must have some evidence of
localization of the origin of the seizure.
These may be apparent from the history
of the attack, observation of the sei-
zure, neurologic findings, electroencephalo-
graphic examination either spontaneous
or activated, or roentgenograms of the
skull with or without air injection. The
localization so determined will indicate the
proper placement of the osteoplastic flap.

Anesthesia. Local infiltration of the
scalp along the proposed incision is the
anesthesia of choice, because it does not
interfere with the cortical activity nor the
state of consciousness of the patient who
can report subjective experiences during
the cortical exploration. Morphine may
be used to allay restlessness or anxiety.

Operative Technic. A large osteoplastic
flap is turned down to expose the epilepto-

Fig. 9. Sketch to show the method of application of cortical electrodes.

genic focus. Occasionally the type of skin incision may be dictated by previous scalp wounds. A tantalum plate may have to be removed. In most cases the entire procedure can be done in one stage. In certain instances, however, where the lesion lies close to the midline necessitating exposure of the longitudinal sinus it may be wise to perform the operation in two stages. In the first stage the bone flap is turned down and tantalum foil or plate placed over the longitudinal sinus. This will decrease the likelihood of serious bleeding should the patient have a seizure as the result of cortical stimulation.

The dura mater must be reflected carefully to avoid tearing subdural adhesions. Sharp dissection is advisable to cut meningocerebral scars. If there is an extensive adhesion, it may be better to open the dura mater only at the margin of the lesion where the epileptogenic focus is thought to lie.

When the lesion and surrounding cortex have been exposed, the cortex is systematically explored by electrocorticography and electrical stimulation.

A special electrode holder* is clamped to the margin of the bone. Ten electrodes are placed on the exposed cortex to record the spontaneous and induced activity. (Fig. 9.)

Penfield and Jasper[1] describe the localized electrocorticographic alterations in focal epilepsy as consisting of large spikes or sharp waves appearing in random sequence against a background of lower amplitude activity arising from the border zone between the lesion and normal cortex. In fourteen of thirty-nine cases examined by electrocorticography Walker et al.[2] found such focal spontaneous epileptic activity confined to a portion of one gyrus. (Fig. 10.) Occasionally such a

* The electrode holder used in this Clinic was designed and made by Dr. G. Marshall.
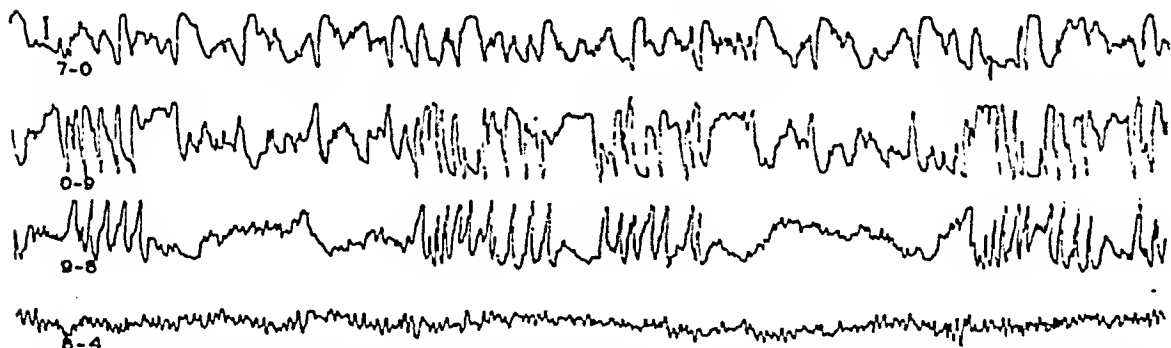
FIG. 10. Electrocorticogram from the right inferior frontal region of a patient suffering from post-traumatic epilepsy, the result of a severe penetrating wound of the head. The spiking activity at both points 0 and 9 is well shown. The horizontal line at the base indicates an interval of one second.
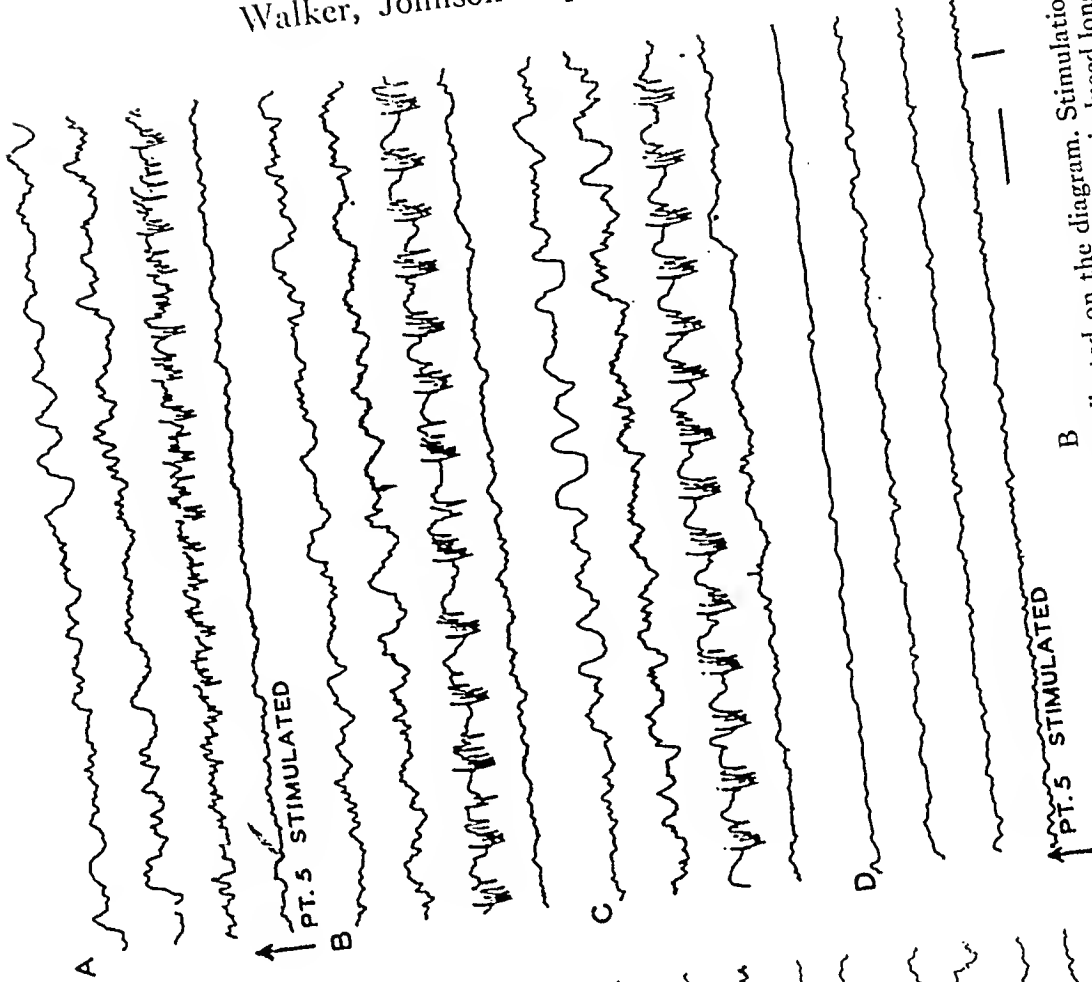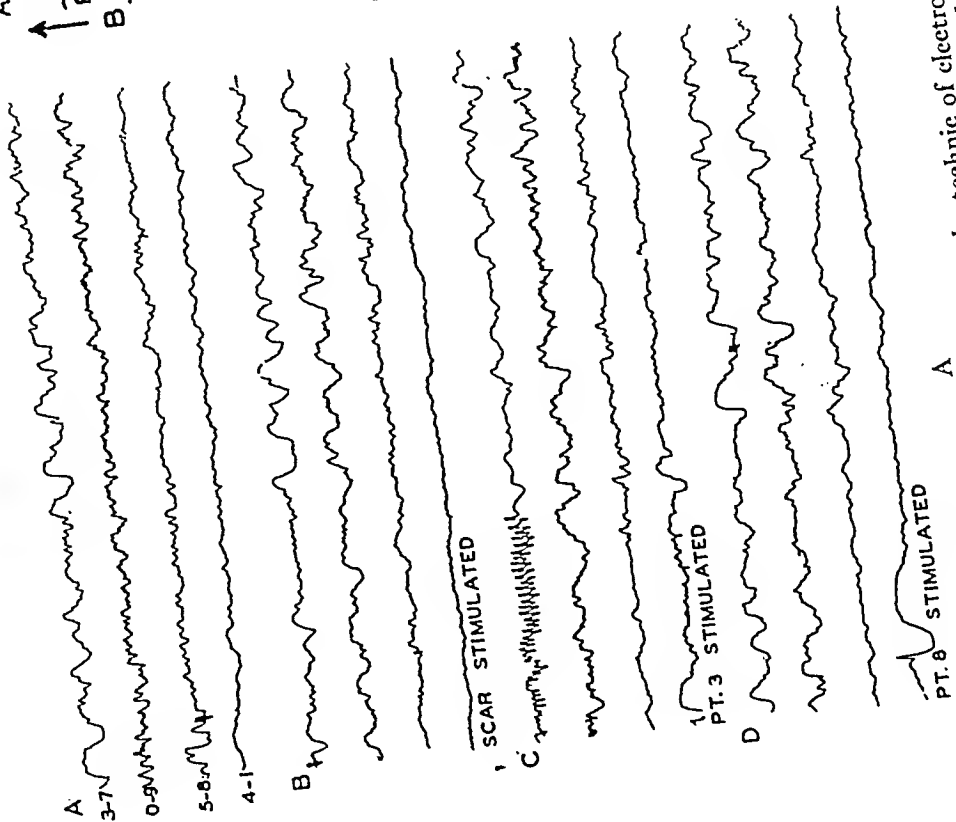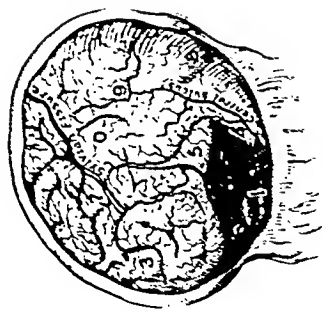
Fig. 11. A, electrocorticograms showing the technic of electroactivation. The electrodes are placed as indicated on the diagram. Stimulation of point 5 induced little alteration. B, stimulation of point 5 induced long-lasting localized after-discharge. After removal of the cortex of the triangular gyrus and the area beneath point 5, stimulation no longer caused an after-discharge. The horizontal line at the base indicates an interval of one second and the vertical line a calibration of 50 microvolts.
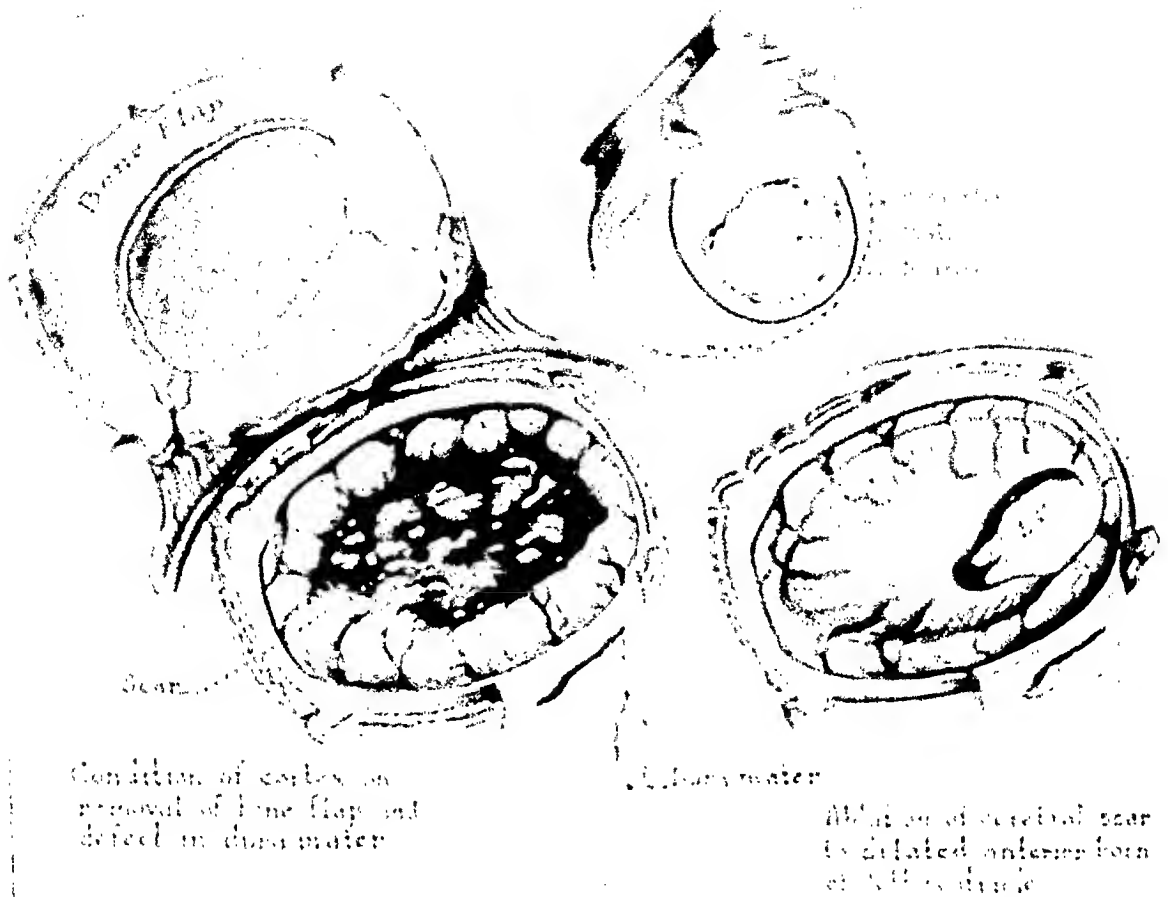
Fig. 12. Sketch to illustrate the surgical removal of a meningocerebral scar to the ventricle.

focus was found in normal appearing convolutions some distance from the scar.

The cerebral cortex is then explored using a sine wave current from 1.5 to 2.5 volts for five seconds. The electrical activity of the cerebral cortex, the motor responses and the patient's subjective sensation at the time of stimulation are noted. If no change is observed in the electrocorticogram after a minute interval, another point is stimulated and so on until the entire cortex has been mapped. An after-discharge lasting for a few seconds at the point stimulated is considered within physiologic limits. However, generally an area of cortex adjacent to the scar can be found which on stimulation induces a long lasting spiky after-discharge. This epileptic discharge has been seen to persist for as long as twenty-eight minutes. (Fig. 11.) In many cases the sensory aura which

precede the patient's spontaneous attacks occur with this after-discharge. Occasionally the electrocorticographic attack progresses to a clinical seizure. But in some cases the individual does not have any motor or sensory concomitants during the electrocorticographic discharge.

With this technic the likelihood of the patient developing a severe seizure is much less than with the use of uncontrolled electrical stimulation. There is evidence to suggest that the focus found by electro-activated electrocorticography and that localized by electrical stimulation alone are not quite identical. On many occasions the after-discharge is present for several minutes and changing in character before the patient notices the aura of his attack. Frequently the latter is associated with decreased electrocorticographic activity. This suggests that the

Temporal

Frontal

Occipital

pia mater
and
arachnoid

SUCKER

Midline

Subpial resection -
shaded area
indicates cortex
removed.

Cross-section showing
subpial resection. Heavy
dark line indicates part of
cortex removed.
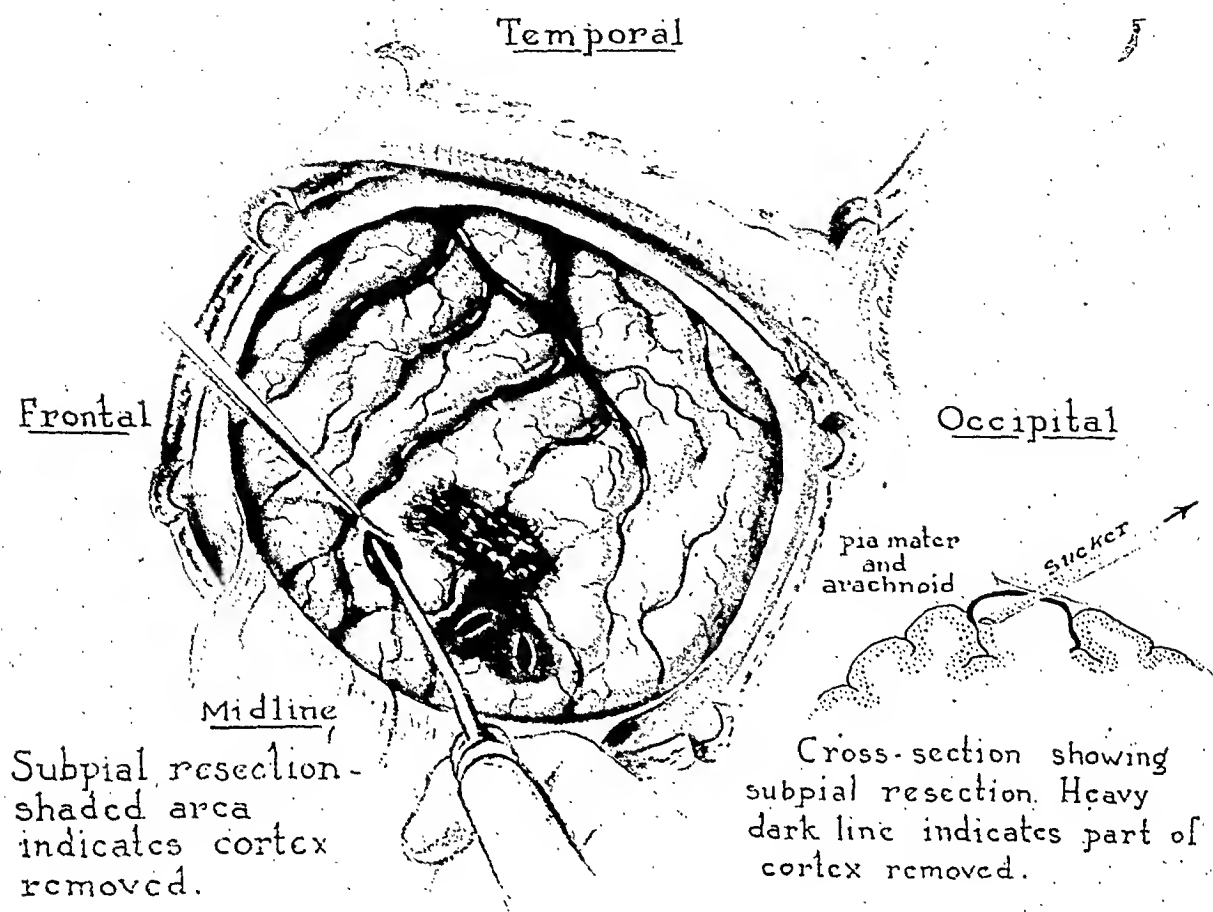
FIG. 13. Sketch to show the technic of subpial resection of an epileptogenic focus adjacent to a meningo-cerebral scar.

electrocorticographic focus is a primary focus which fires a secondary focus initiating the aura of the attack.

By rearrangement of the cortical electrodes and push-pull recording the epileptogenic focus can be delimited. It is usually adjacent to the cerebral scar but occasionally in a gyrus slightly removed. Multiple foci are occasionally present and a focus may migrate rarely from one cortical area to another. In most instances, however, the focus is constant and the typical after-discharge can be reproduced time after time.

In those cases in which the clinical aura and electrocorticographic manifestations of an attack are induced it seems probable that the stimulated focus is responsible for the patient's spontaneous attacks. In those cases in which only the

electrocorticographic epileptic manifestations can be elicited, the evidence that the point stimulated is the origin of the attacks is not conclusive, even though there is no doubt that such areas are functioning abnormally and are potentially if not actually epileptogenic.

The epileptic focus having been located and its extent determined, it is removed by one of several technics, excision of the focus and the scar to the ventricle, removal of the focus and the scar to normal white matter or subpial resection of the focus.[13,22,50] Subpial resection of the focus is done by making an incision in an avascular portion of the pia arachnoid and ablating the cortex by subpial dissection and suction. The involved gyrus is removed completely from trough to trough. (Figs. 12, 13 and 14.)
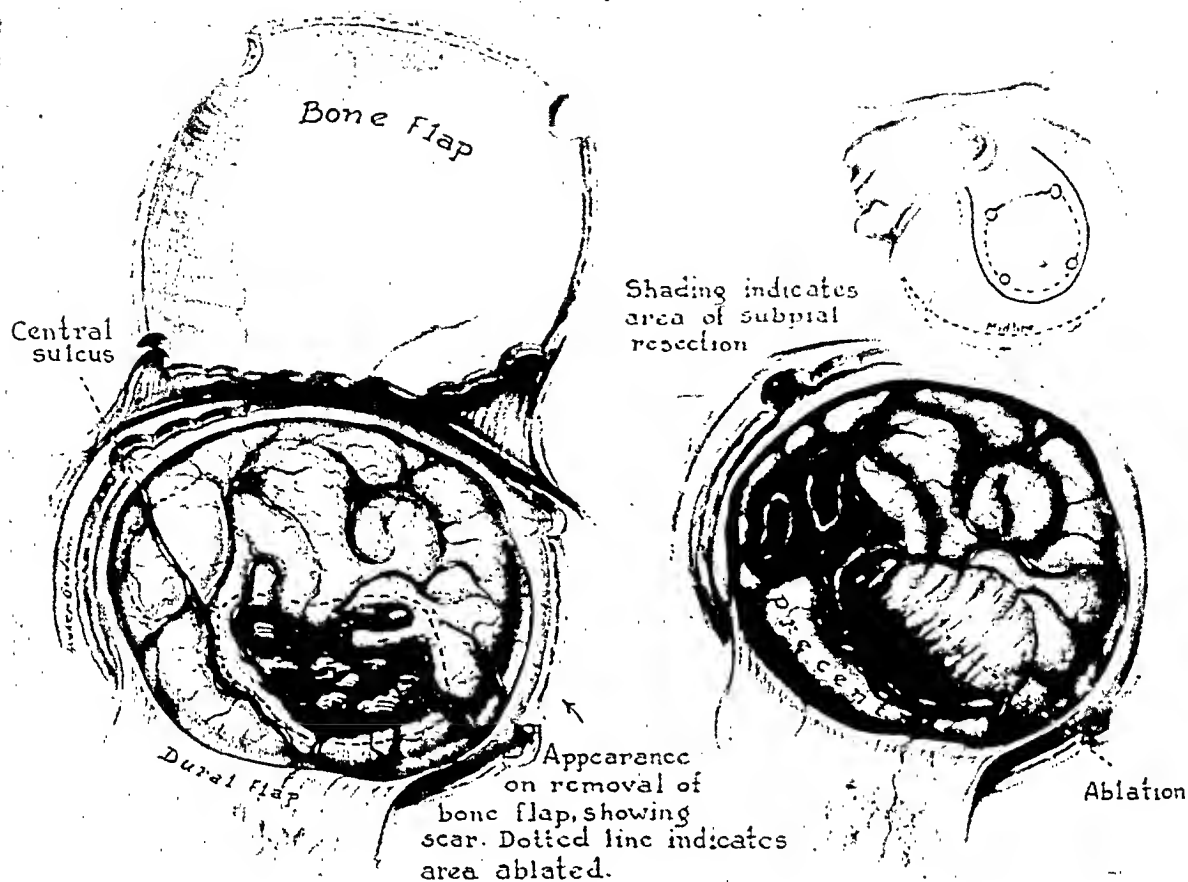
FIG. 14.

The decision as to which of the several technics to use will depend on evaluation of the individual case. It is not desirable to leave the patient with a more severe neurologic deficit than he had previously. If the scar is large and adjacent to the motor area, extensive resection may aggravate a paralysis or aphasia. The danger of complications is lessened if the ventricle is not opened. In the occasional case in which a clear cut focus cannot be demonstrated, it is probably well to excise the scar and entire surrounding epileptogenic zone. Irrespective of which procedure is carried out, the blood supply to the remaining cerebral cortex is disturbed as little as possible.

Following removal of the focus the electrodes may be placed upon the adjacent cortex to determine any remaining epileptogenic tissue. In some cases before all convulsive activity can be eliminated a rather extensive subpial dissection of several gyri running into the scar must be carried out. During the ablation, speech and motor power are checked frequently.

Postoperative Medical Therapy. After any operative procedure for the relief of epilepsy the medical management, especially anticonvulsant drug therapy, must be continued for a period of several years. If no further attacks occur, the medication may be decreased at the end of two years and gradually eliminated over another two-year period. If seizures occur, the medication may be augmented or changed. The patient's social and economic rehabilitation will be greatly facilitated if, through vocational guidance, an occupation, suitable to his or her physical, mental and psychologic status, can be found. Continued guidance may be necessary to effect a satisfactory adjustment to all of these factors.

Results. The largest series of patients treated by surgical extirpations of focal epileptogenic lesions has been reported by Penfield and Erickson.[39] During a ten-year period 165 cases were operated upon with a mortality of 4.2 per cent. In the group of patients with excision of a meningocerebral cicatrix, 22.5 per cent were completely free from attacks, 64.5 per cent were at least 50 per cent improved, 11 per cent unchanged and 2 per cent worse. In the group of patients with a cerebral cicatrix, 19 per cent were completely free from attacks, 49 per cent were improved, 30 per cent unchanged and 2 per cent worse. In a group of 25 patients having negative explorations, 2.5 per cent were completely free from attacks, 22.5 per cent improved, 69 per cent unchanged and 6 per cent worse. At the present time it would seem that cortical excisions may offer help to a certain group of patients not benefitted by medical management.

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FRACTURES AND DISLOCATIONS OF THE SPINE*

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ONE of the most difficult and perplexing problems confronting a physician is the management of a patient with a spinal injury. This is particularly true when the spinal cord is injured or endangered. Not only does it involve the immediate treatment of a potentially dangerous fracture but also the care of a profoundly disturbed physiology. So altered are body mechanisms, it is only by constant effort and exchange of professional ideas that optimum anatomic and physiologic restitution is possible.

Spinal injuries are caused by direct or indirect trauma. Direct injuries result from a heavy object striking the spine, a crushing force applied to the spine or penetration by high velocity missiles. The more common vertebral injuries result from indirect trauma as caused by falls or diving accidents in which strong force is applied along the axis of the spine crushing or dislocating vertebrae.

Dependent upon the intensity, duration and direction of the force applied to the spinal column, extreme flexion, extension or rotation results in ligamentous tears, fractures or dislocations of vertebrae. Probably the fracture most frequently encountered results from falls or automobile accidents in which the spine is abruptly flexed by a tremendous force. Less commonly, fractures due to extreme extension of the spine occur. Although fractures with or without dislocation may occur at any level, they favor the levels of greatest vertebral mobility—the lower cervical region (C_5, C_6 and C_7) and the thoracolumbar junction (D_{12} and L_1). Aside from actual bone displacement, tearing of the vertebral ligaments or cartilages with

their impingement upon the spinal cord or nerve roots may result.

Whatever the mechanism of indirect injuries, the resulting deformities of the vertebral canal are remarkably constant:

1. The vertebral canal is narrowed in its anterior posterior diameter by dislocation of a vertebra with or without fracture of the body, pedicles or laminae. Most frequently this type injury is encountered in the low cervical region where spinal mobility is greatest. It is with fracture dislocations that the most severe cord injuries occur. Conversely there may be considerable narrowing of the canal in the cervical spine without cord injury for in this region the canal is large.

2. Crushing injuries of the vertebrae with wedging of the bodies and posterior displacement of fragments into the vertebral canal are commonly seen in the thoracic region. Any portion of the vertebral arch, however, may be fractured and displaced to impinge upon the cord.

3. Complete obliteration of the spinal canal is found in some of the more serious cases. This is not uncommon in extreme fracture-dislocations of the twelfth dorsal and first lumbar vertebrae due to violence to the shoulders.

4. Depending upon the severity of trauma, the degree of flexion, extension or torsion of the spine, a variety of vertebral fractures may take place with a varying degree of bony or ligamentous encroachment upon the vertebral canal.

In any series of spinal cord injuries, a variety of neurologic disturbances is manifest depending upon the actual amount of cord damage. Edema of the cord may develop at the site of injury and extend

several segments above and below. This may be sufficient to interrupt additional cord function for a short period. As it abates, function returns, at least in part, except in those cases showing evidence of a complete transverse lesion.

The impact of displaced bone upon the cord frequently produces diffuse hemorrhages as well as perivascular extravasations particularly in the gray matter. At times, collections of blood may occur in the subarachnoid, subdural or extradural space. With severe fracture-dislocations, laceration and even masceration of the cord is common.

CLINICAL PICTURE OF CORD LESIONS

Complete Transverse. Immediately following a transverse cord lesion from severe contusion or laceration, there is complete motor, sensory and reflex loss below the level of the lesion. If spinal roots above the site of bone injury are involved, further sensory and motor loss are evident. The urinary bladder distends and unless relieved by decompression, incontinence results. Intestinal peristalsis is diminished and obstipation is present. All voluntary bowel control is lost. Both pilomotor and sudomotor activity below the level of the lesion are lost. However, these functions often return to an exaggerated degree within a few weeks. Trophic skin changes may readily appear over bony prominences unless these parts are well protected. It is not unusual to see bullae over the sacrum and buttocks within a few hours after a complete cord lesion. Edema of the lower extremities and back is common. Depending upon the level of the lesion, numerous clinical pictures may result.

Lesions involving the upper four cervical segments produce quadriplegia with a sensory loss extending up to the clavicles. Paralysis of the diaphragm results from lesions at C_3 or above and breathing is possible only through the use of the accessory muscles of respiration. As in all cord lesions above the first thoracic vertebra, miosis from paralysis of the dilator of the pupil may be seen. Hyperthermia, the etiology of which has not been completely explained, is common. Mucus accumulates in the respiratory passages and aspiration is necessary. These are grave lesions and it is seldom that the patient survives for more than twenty-four to forty-eight hours. With complete cord transsections in the lower cervical spine, there is paralysis of the lower extremities and weakness of the upper extremities depending upon the level of the cord lesion. The hand grips are always weakened. Breathing is abdominal in type as the intercostal muscles are paralyzed. The sensory level is at the third or fourth ribs anteriorly and sensation in all forms is abolished below this level.

Crushing fractures of the thoracic spine (below the first thoracic vertebra) in peacetime usually result from severe flexion or extension injuries. The already small vertebral canal is further diminished in size by displaced fragments or by dislocation of vertebrae. The upper extremities are unaffected while all sensation and motor power is lost below the level of injury. With fractures of the lower thoracic and first lumbar vertebrae because of the close proximity of cord segments, it is rare to have but one segment involved. More frequently, several segments of the cord and portions of the cauda equina are crushed. By determining the level of sensation, motor power and reflexes, the upper level of cord injury may be ascertained.

With fractures below the first lumbar vertebra, it is the cauda equina which may be involved. As the cauda equina is made up of peripheral nerves, the prognosis is much more favorable than with injuries to the spinal cord. The extent of nerve injury is evident by the motor and sensory loss in the lower extremities.

Incomplete. With injury to but a portion of the grey or white matter of the cord at any one level, a variety of neurologic disturbances dependent upon the extent and severity of cord trauma occur. When in the cervical region, a partial to

complete paralysis of the upper and lower extremities on one side and loss of pain and temperature sensation of the contralateral extremities may be seen (Brown-Sequard Syndrome). The reflexes are inconstant, being diminished or absent in the paralyzed extremities. Another common clinical picture is a partial to complete bilateral paralysis of the upper and lower extremities, dependent upon the level of injury with preservation of some appreciation of external stimuli applied to the paralyzed area. Reflexes may or may not be elicited. Incomplete cord lesions in the thoracic region (below D-1) may present similar pictures except the upper extremities are uninvolved and the transition from absent or diminished to normal sensation occurs from the inguinal ligaments to about the third or fourth ribs.

Incomplete lesions of the lumbar cord (L-1) produce motor and sensory impairment limited to the lower extremities together with sphincteric and visceral disturbances which accompany almost all severe injuries of the spinal cord and cauda equina. There is partial to complete paralysis of one or both lower extremities. Knee and ankle jerks are usually absent. Anesthesia of the stocking type may be found associated with the more severe injuries.

Incomplete lesions of the cauda equina result in paresis to paralysis of individual muscle groups depending upon the number of nerve roots involved. Sensation is impaired or lost in the dermatomes supplied by these nerves. Since motor components of the cauda equina have power of regeneration, the prognosis for at least some recovery is much more favorable than in cases of spinal cord injury.

TREATMENT

The immediate treatment of a patient with or suspected of having a fracture of the spine with or without neural involvement is his careful transportation to a hospital, due caution being exercised against any movement of the spine. Too frequently these patients are picked up at the scene of accident and rushed to a hospital in the first available conveyance, no thought being given to the possibility of a spinal injury. Unfortunately, some patients have also been rendered unconscious and the possibility of cord injury is not suggested. Perhaps the best method of transporting any patient with a possible vertebral fracture is on a board or similar flat, unbending surface. As in any injury, all available detail regarding the production of the injury and especially the rapidity of onset of neurologic disturbance should be obtained. When first seen, many patients are in shock which is treated by the usual methods. Further treatment should be guided by the results of: (1) neurological examination, (2) x-ray studies of the spine and (3) lumbar puncture.

Neurologic examination should be carried out in detail and recorded for future reference. This is especially helpful when incomplete lesions of the spinal cord or cauda equina exist, for then any progression or recession of signs may be followed. Much, too, can be learned of the prognosis, for with a history of sudden onset of paralysis following injury with neurologic evidence of a complete physiological transsection of the cord, there is little or no hope for functional improvement especially if none is evident after a few days. In general, the more cephalad the neural injury, the more serious the injury and higher the mortality. In transverse cervical cord lesions, considerable respiratory dysfunction results and even with the best of care a large percentage of these patients succumb within ten days.

X-ray studies of the spine should be conducted with great care to avoid undue motion or handling of the patient. With suitable films, the extent of vertebral damage, displacement of bone fragments and their encroachment on the spinal cord can be ascertained. Lateral x-ray views of the spine are the most valuable views. These studies are extremely helpful in estimating neural damage, guiding treat-

ment and evaluating its results. At times, no x-ray evidence of fracture is found and spinal cord injury is believed to be due to vertebral luxation with recoil to its original position or to massive extrusion of a nucleus pulposus. This is not an infrequent finding in the cervical region. Fractures and fracture-dislocations of the sixth and seventh vertebrae are frequently overlooked due to technical difficulties in obtaining suitable delineation of the lowermost part of the cervical spine.

Lumbar puncture is carried out as soon as the condition of the patient permits, due care being taken not to disturb the spine. The presence of bloody spinal fluid usually indicates a severe injury of the spinal cord or cauda equina. However, its absence does not lead to a more favorable prognosis when the neurologic examination indicates a severe lesion. The patency of the subarachnoid space is of importance for the course of subsequent treatment is often guided by it as emphasized by Coleman.[1] By compression of the jugular veins there is a prompt rise in cerebrospinal fluid pressure which is transmitted throughout the subarachnoid spaces. Obliteration of the subarachnoid spaces of the spinal canal prevents transmission of pressure changes below the level of the block when compression of the jugular veins is carried out. Thus, with a lumbar puncture needle in place and connected to a manometer, if no rise of cerebrospinal fluid pressure occurs with jugular compression, complete obstruction of the subarchnoid spaces exist (positive Queckenstedt test). Should there be a slow rise of the fluid level on compression of the veins followed by a slow fall on release, an incomplete block is said to exist. Should there be evidence of pressure on the cord or cauda equina, treatment in most cases should be directed toward its removal.

The Queckenstedt test is a valuable adjunct to the treatment of spinal injuries but like many tests, when accepted at face value, it can lead to hasty or inadequate treatment. A block may persist for several days or longer following reduction of a dislocation or even after laminectomy when, in most cases, it is known that the spinal cord or cauda equina is not seriously compressed. This probably results from torn membranes at the site of injury or from the subarachnoid space having been sealed off due to pre-existing pressure. It is seldom, therefore, that additional treatment is necessary unless paralyses increase or fail to subside as would be expected. Conversely, a negative Queckenstedt should not always be accepted as a contraindication for laminectomy. Not infrequently, fragments of bone from vertebral bodies or laminae exert pressure against the spinal cord or cauda equina without producing obstruction to the flow of cerebrospinal fluid. It is not our purpose to minimize the importance of the Queckenstedt test, but we do believe that it is not an infallible guide to treatment.

From the standpoint of treatment and prognosis, the cervical, thoracic and lumbar regions of the spine should be discussed separately as each presents individual problems.

Treatment of Cervical Injuries. Injuries of the cervical spine endanger life and function to a greater extent than those at other levels. Furthermore, due to the extreme mobility of the cervical spine, augmentation of spinal cord compression is not uncommon. Therefore, these injuries must receive immediate and adequate treatment.

Fortunately, the cervical spine lends itself admirably to traction. By means of gentle, skeletal traction most dislocations can be reduced within one or two hours, or sooner if heavy traction is applied. We have always preferred the former method because it is less injurious to the brachial plexuses and spinal ligaments. Although an hour or more may be required for complete reduction, decompression of the spinal cord begins almost immediately after the application of effective traction.

Perhaps there are a few circumstances, mostly theoretic, which would cause one

to discontinue traction and employ laminectomy. But during the fourteen years which have elapsed since we (W. G. C.) introduced skeletal traction,[2] we have not found any indication for laminectomy in the cervical region. In the past, massive extrusions of the nucleus pulposus, especially in the cervical region, probably have been overlooked. Perhaps some of the patients diagnosed as having had a recoiling dislocation were paralyzed as a result of a herniated nucleus pulposus. In our experience, all such cases without x-ray evidence of bone injury were completely paralyzed. Therefore, laminectomy if indicated would not have altered the prognosis. It seems certain, however, that the incompletely paralyzed patient should be given the benefit of exploratory laminectomy if he presents little or no x-ray evidence of bone injury together with a persistent block.

Uncomplicated anterior compression fractures of the cervical spine seldom require more than immobilization in plaster or a good adjustable brace for approximately three or four months. The more serious compression fractures, especially those with fragments jutting into the spinal canal should, in our opinion, be treated by means of skeletal traction for a period of at least four to six weeks before the application of a fixed support. In some cases, we have used traction for a shorter period but have kept the patients in bed for several weeks after the application of plaster. In our opinion, casts and other appliances are completely inadequate when strong support is required.

We have used small skull tongs for twelve years and have found them entirely satisfactory regardless of the pull required. The use of this instrument has been described by one of us (W. G. C.) in several previous publications.[3-6] Failures, usually due to the instrument slipping out, result from improper application, failure to tighten the clamp when the instrument is loose or from the use of defective tongs.* Other methods of applying skeletal traction have been described.[7-10] These consist of using either large tongs which prevent the patient from being turned on his side, or wires through the skull which, while effective, require some knowledge of neurologic surgery.

To obtain perfect or satisfactory alignment of the cervical spine is comparatively easy but to maintain it may be difficult. Dislocations of the atlas or axis almost invariably recur even after being held in a corrected position by means of traction for several weeks. Therefore, we believe that fusion, such as that described by Cone and Turner,[11] is indicated in these cases and that it should be performed without undue delay. Dislocations lower in the cervical spine are often associated with fractures of the pedicles which make it difficult to hold the vertebrae in alignment after the removal of traction. In recent years, we have continued traction in such cases from four to six weeks before applying plaster and allowing patients out of bed. This plan of treatment has been much more satisfactory than the earlier one which permitted early ambulation and we have seldom found it necessary to resort to spinal fusion. Many of the potentially ambulatory patients with dislocations below C-2 and with intact pedicles require only a short period of hospitalization. Usually, the deformity can be corrected and plaster applied within twenty-four hours. All of these patients, particularly those who are up and about, should be checked by x-ray examination at least once a week for a month or until it is determined that the corrected position will be maintained.

* During the war, it became necessary to have a large percentage of the Crutchfield tongs made by subcontractors, and, as a result, these instruments were defective. Instead of opening to a minimum of 10 cm. (between points with the instrument open), many had a spread of only 6.75 cm. which was inadequate except for very narrow skulls. This error has been corrected and the points have been improved as a further safeguard against excessive penetration of the skull.

Treatment of Thoracic Injuries. In the thoracic spine, compression fractures are the rule and the incidence of severe spinal cord injury is high, due to the comparatively small canal in this part of the spine. Because of its attachments to the rather rigid thoracic cage, the thoracic spine is seldom the site of dislocations. When they do occur, they are the result of extreme violence and almost invariably the spinal cord is hopelessly destroyed.

Simple compression fractures associated with little or no cord injury are mainly orthopedic problems. Hyperextension on a Bradford frame, followed by the application of a plaster jacket, usually is sufficient to bring about an entirely satisfactory result.

Fractures and dislocations associated with a complete transverse cord lesion require little in the way of specific treatment. Laminectomy is useless but it may be performed in selected cases to give the patient the benefit of a doubt especially when a spinal subarachnoid block persists. Also, laminectomy or removal of spinous processes may be indicated for the elimination of a bony prominence over which a pressure sore is apt to develop.

Laminectomy is the procedure of choice for decompression of the spinal canal in the thoracic and upper lumbar spine (through L-1). Laminectomy is indicated and it should be performed as soon as the patient's general condition will permit if he presents evidence of a severe but incomplete spinal cord lesion together with a subarachnoid block. When the spinal cord injury is slight and x-ray evidence of cord compression is lacking, immediate operation may not be altogether necessary even though a block exists. Unusual delay in neurologic recovery or certainly increasing paralysis would be an indication for laminectomy in such cases.

Treatment of Lumbar Injuries. The spinal cord extends to the lower part of the first lumbar vertebra; therefore, when discussing injuries of the lumbar spine, which generally offer a favorable prognosis, one should exclude the first lumbar vertebra for lesions at this level present the same problems as those in the thoracic spine. The lumbar canal contains the cauda equina which is composed of motor and sensory peripheral nerve roots. The motor roots have power of regeneration which accounts for the return of at least some neurologic function which occurs in the majority of patients with paralyses resulting from fractures and dislocations of the lumbar spine.

Simple compression fractures of one or more bodies of lumbar vertebrae, resulting from acute hyperflexion of the spine, are common and as a rule only hyperextension and the application of a cast are required. These injuries seldom disturb or endanger the cauda equina.

Severe compression fractures with fragments of bone displaced posteriorly often are associated with paralyses of the lower extremities, varying in degree. Usually, a spinal subarachnoid block is found in such cases. Decompression of the cauda equina by means of laminectomy followed immediately or later with spinal fusion especially if the patient is expected to recover motor function, is the procedure of choice in most of these cases. However, in certain cases showing only moderate posterior displacement of bone fragments, gradual hyperextension without laminectomy is justifiable. When this is done, constant attention should be given to the patient's neurologic status and the procedure must be immediately discontinued if increasing neurologic dysfunction is observed. If there is dislocation with or without fracture, this or any other part of the spine should not be hyperextended without first bringing about a reduction of the dislocation.

Occasionally, in the lumbar spine one encounters a complete dislocation with over-riding of the two ends. Needless to say, such injuries are incompatible with return of neurologic function regardless of the type of treatment employed. Many of

these patients live indefinitely and their neurologic deficit is no greater than in other patients with a good weight-bearing spine. Therefore, we have practiced open reduction in such cases to provide a weight-bearing spine and to remove bony prominences which endanger the overlying skin. Operation, of course, is always postponed until the patient's general physical condition is satisfactory.

GENERAL CARE OF THE PATIENT

The general care of the paralyzed patient should be well understood but so often it is woefully lacking in one or more details. This is understandable when one realizes how much constant nursing care and supervision these patients require especially during the first few weeks following injury. When pressure sores and urinary complications are prevented, primary hospitalization usually should not exceed two to four weeks. Then the patient can be transferred to his home, to a rehabilitation center or to a hospital equipped to take care of his prolonged or permanent needs. If complications especially pressure sores develop, the patient may not be moved for many months during which time both the patient and hospital sustain an enormous economic loss.

It is a common observation that private patients who can afford special nursing care almost never develop pressure sores, whereas ward patients almost invariably do. To protect patients and to utilize hospital beds to the fullest extent, our hospital authorities were made to realize the wisdom of providing special nursing care for all patients regardless of their financial status. During the several months that this plan has been in effect, no patient has developed a pressure sore and our paraplegics have remained on the service for a comparatively short period of time.

Loss of bladder and bowel control is one of the most serious and disturbing effects of spinal injuries, and unfortunately methods for restoring anything like normal or satisfactory control in the severely paralyzed patients have not been devised.

Management of the paralyzed urinary bladder varies in different clinics, but the objective is essentially the same, namely, to establish adequate drainage with minimum urinary sepsis until automatic function develops. The plan we use and find as satisfactory as any is as follows: An indwelling catheter is used in females. The same is used in males, for several days, until the prognosis as to life and function is determined. If the male patient has a complete or very severe cord lesion, suprapubic drainage is instituted. If the cord lesion is such that voluntary control may return within perhaps four to six weeks, tidal drainage such as that described by Munro[12] is employed. In both males and females mechanical drainage is discontinued when automatic bladder function appears, usually within four to twelve weeks. This is present when there is intermittent expulsion of urine through the urethra or around the tube. With proper timing and by exerting pressure over the lower abdomen, some of these patients get along reasonably well whereas others are less fortunate and must depend on using a receptacle strapped to the body or thigh. In all cases attacks of urinary sepsis are common and the attention of a urologist is indispensable.

Usually by regulating the diet together with the use of mild laxatives, such as mineral oil, the bowel contents can be kept at a semi-solid consistency and can be removed by enema. Unless this is done, either fecal impactions develop or the stools become so soft that soiling and maceration of the skin become very troublesome.

One of our most important problems has been the after-care of permanently paralyzed patients. With the establishment of paraplegic centers for veterans, it is now possible for many patients, primarily treated in civilian hospitals, to be transferred to these centers where specialized care, including vocational training, is

provided. Until recently the civilian, without a service record, had no place to go. Now, some states, of which ours is one, are opening rehabilitation centers which should offer some hope and encouragement for these unfortunate patients.

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SPONDYLOLISTHESIS, though primarily an orthopedic problem, may progress to such a degree that changes in the nervous system require neurosurgical treatment. The most commonly reported involvement of the nervous sytem consists of compression of the cauda equina by the narrowed canal, resulting in varying degrees of sensory and motor loss in the sacral segments.

PREFRONTAL LOBOTOMY*

INDICATIONS AND RESULTS IN SCHIZOPHRENIA

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IT has been reliably estimated that approximately 40,000 new patients are admitted to mental hospitals each year with schizophrenia and that about a quarter of a million hospital beds in this country are constantly occupied by schizophrenics. Probably around no other mental illness has there been so much controversy and so much difference of opinion concerning its origin, etiology, symptom formation and treatment.[1] This has led one investigator to ask, "Is schizophrenia an adaptation or is it a disease?" How a patient with this condition can live on for years if not for decades and then come to necropsy with a brain that shows no indications of disease either grossly or microscopically is a challenging problem. Our approach to the problem has been neurophysiologic and our attitude has been pragmatic.[2]

Our first lobotomies for schizophrenia were performed in 1936 with immediate relief of symptoms, but within a few months several had relapsed. Some of these were reoperated upon but end results were modest. About the time our patients began to relapse, enthusiastic reports upon the effect of insulin shock therapy began to appear in the literature so we discontinued the operative treatment. Then gradually, as follow-up studies revealed that the early reports on shock therapy had been overly optimistic, we cautiously resumed the surgical treatment with an improved technic. When our book, Psychosurgery, was published in 1942[3] there were only twelve schizophrenics in the series; we now have approximately 250. It was about this time that Strecker, Palmer and Grant[4] showed that excellent results could be achieved in the chronically disturbed schizophrenics. This work was followed up on a large series of cases by Schrader[5] and others, so we followed their lead and achieved comparable success.

When a patient with schizophrenia is being considered for lobotomy, a number of questions must be answered: What symptoms are present which can be relieved? Is there any other method available to the individual which offers a reasonable hope of recovery? Is the condition serious enough to risk a major brain operation and all this implies? Will the family be satisfied with the modest result or do they anticipate a miracle? Can the family care for the patient during the convalescent period after he leaves the hospital?

Obviously not all schizophrenics will be improved by psychosurgery. The best results can be secured in those who show evidence of emotional tension. The emotional component can be judged by the severity of the complaints, the disturbances in behavior and the deviations in autonomic balance. When there is no history of outbursts of temper and assaultiveness, and the patient sits quietly in a corner during the interview listening with indifference to the voices, and examination reveals normal pupils, pulse and peripheral circulation, then the fight is over. It is too late for psychosurgery because a deteriorated precox behaves the same before and after prefrontal lobotomy.

THE PRECISION METHOD OF OPERATION

Prefrontal lobotomy is performed through burr holes made through the coronal suture and in the standard operation the path-

ways are cut in the plane of the suture down to the sphenoidal ridge. In the more severe cases of longer duration the pathways are severed 6 to 12 mm. posterior to the sphenoidal ridge. Operation severs the thalamofrontal pathway and specimens show degenerative changes in the medial dorsal nucleus of the thalamus. Severing causes a reduction of disappearance of the emotional feeling tone that attaches itself to the ideas elaborated by the frontal lobes. The surgical problem is to place the incisions far enough posteriorly to reduce the charge to the point where the ideas no longer interfere with the patient's ability to work, yet not too far. Each millimeter of tissue behind this plane that must be sacrificed reduces the ability of the patient to adapt himself in a social environment.

Standard prefrontal lobotomy was performed in 63 per cent of the patients and the radical operation was used in 37 per cent. Twelve per cent required two or more operations to relieve the symptoms.

Nearly all of the patients who are referred for prefrontal lobotomy have had previous psychiatric care and shock therapy. Most have received electroshock and many have had insulin and metrazol. The number of shock treatments has ranged from 15 or 20 to over 200. The majority of patients have been hospitalized so it is obvious that they are disabled. A small minority come to us directly and these have received psychotherapy and electroshock. It is our opinion that most patients in whom shock therapy is effective begin to show definite improvement after six to ten treatments. If no improvement occurs after this and if there is an intense suicidal drive, institutionalization or lobotomy is recommended. Although a recent report from England covering 1,000 cases indicates that the results are much better when prefrontal lobotomy is done early in the course of the disease, we rarely employ it unless the symptoms have been present more than two years. If shock therapy gives a temporarily good result we are inclined to believe that lobotomy will give a prolonged good result. However, the fact that shock therapy fails does not mean that lobotomy will also fail. If an individual is not facing disability or suicide, a lobotomy is not indicated. The fact that a person exhibits a schizophrenic reaction does not constitute an indication for operation. As a matter of fact, when a whole family comes to the office for an interview, it is not always easy to pick the patient out of the group. However, we operate on the one who is disabled rather than the one with the craziest ideas.

In private practice it is almost as important to select the family as to select the patient, because they play such an important rôle in the convalescent care. They may have read an account of the operation or may have heard of some patient in the same institution who was operated upon with an excellent result. Naturally, they expect the same, not realizing that their son broke down under different circumstances and has now reached a stage of deterioration so that the most that can be hoped for is that he can live at home. They must be cautioned against expecting a full return to normal. It is our opinion that a patient who can go directly home ten days after operation has a much better chance of achieving a satisfactory social adjustment than one who must return to even a good mental institution. At home all kinds of pressure are brought to bear to make him behave normally. In an institution he is treated like a patient. If he wets or soils himself, someone cleans him up and then most likely he is transferred to a back ward where other patients are behaving in a similar manner and he will not be noticed. The lobotomized patient is like a child and the family is presented with an opportunity to train him over again. If he is allowed to develop bad or slovenly habits, the golden opportunity may be lost. It is true that the going may be difficult. Therefore, arrangements must be made prior to operation for some member of the family, preferably a mother, a sister or a brother who can devote his full time to the patient

for a period of at least three months and often six months. In taking the history, we inquire about the size of the house, the members of the family who live there, and what members of the family may be called on for assistance. The fact that some member of the family is willing to pay the hospital, special nurses and surgeon indicates considerable interest in the patient, so this is an immediate advantage.

As our studies in psychosurgery progress, the rôle of the operation itself will gradually fall into its proper perspective. In the beginning, it seemed probable that after cutting certain pathways in the frontal lobes, the patient would use what was left as best he could, and that would be that. It is almost that simple in older individuals with agitated or involutional depression of short duration. They go home a week or ten days after operation with the agitation and feeling of guilt gone, the emotional tension is reduced, they stop worrying about trifles, and if the family does not like the way the place is run, they can do it themselves.

In schizophrenia the problem is not so simple. At first we told the family that improvement might continue for six months after operation, then a year, and now we believe that improvement may continue for three or four years. It has become obvious that the majority of schizophrenics require psychiatric care or at least guidance following lobotomy. Some have been peculiar all of their lives and many developed symptoms at an early age and never had a regular occupation. They do not have a job to go back to like the obsessive compulsives, or housekeeping to resume like the involutionals. They have to develop new interests and these must be in line with their capabilities and opportunities. Instead of taking patients away from psychiatrists, it is more probable that psychosurgery will increase the number in need of psychiatric care as contrasted with custodial care. In mental hospitals lobotomy will reduce the number of special treatment rooms and camisoles, the eu-

phemisms for padded cells and strait jackets, respectively. These patients who were violent, assaultive and altogether unapproachable before operation then become accessible for psychotherapy and occupational therapy.

At the present time, the patient can get more in the home than in most mental hospitals. The Veterans Hospital in Roanoke, Virginia, has started a program of rehabilitation for lobotomized individuals which may prove to be an important contribution. A psychiatrist, a clinical psychologist and a supervising nurse are assigned to the program. They study each patient and try to develop his special interests. One private mental hospital has already started construction of a five story pavilion for psychosurgery. It has facilities for training its patients which includes painting, appreciation of art, architecture, law and what not. With intelligent study of the patients it should prove an ideal place for re-education and rehabilitation. Naturally any such program must take into consideration the deficits produced by the operation, as we have a changed individual to deal with.

As stated earlier, our attitude is pragmatic. . . . We are more concerned with social adjustment and ability to work than with insight and ability to discuss personal problems. With the fantasy life smashed, dreams and hallucinations lose their importance and the individual can turn his attention to things going on around him. He is willing to undertake work commensurate with his ability and is more easily satisfied with his accomplishments. Only 2 per cent of the patients were keeping house before operation and none were employed. Of the 190 schizophrenics who have been followed a year or more after lobotomy, 17 per cent are regularly employed, 15 per cent are employed part time and 7 per cent are keeping house. This means that 39 per cent are usefully employed. An additional 33 per cent are living at home, more or less as drones because of inertia or insufficient relief of symptoms. Only 28 per cent are confined to institutions.

We now have enough information to discuss the prognosis with the family and if they expect no more than we can reasonably offer, then a prefrontal lobotomy may be indicated. Naturally if we think that the patient will be sufficiently relieved to get a job or resume housekeeping, we advise lobotomy. If he has been confined to a hospital for a number of years and the family would be satisfied to have him home if the assaultiveness could be relieved, then lobotomy is indicated. If a patient is so intensely suicidal or homicidal that he is confined most of the time to a strong room and unable to wear clothes or sleep on a bed, lobotomy is indicated even if the chances of leaving the institution are minimal. In fact, these are some of the most gratifying cases. To see an individual changed virtually from an animal in a cage to a person who lives with other patients on an open ward and eats with them in a dining room and walks out in the sunshine again even if he never leaves the institution, seems very worth while.

Since the decision as to whether or not a lobotomy should be performed ultimately depends on the prognosis, it may be well to summarize some of the more important factors which influence the prognosis.

The presence of *emotional tension* is the most important single factor. If it is present, we have something to offer; if it is absent, we have nothing to offer.

The duration of the symptoms and duration of disability are less important than we formerly thought. A follow-up study of 178 cases, followed for a period of one to ten years, reveals that good results may be obtained in 40 per cent and moderate improvement may be expected in an additional 30 per cent. The percentage showing improvement is about the same whether the disability has existed for two years or five years. This does not mean that the time factor is not important. We have in mind a young woman who appeared to be an excellent candidate for prefrontal lobotomy. By the time her family finally reached a decision two years later, she had deteriorated so much that operation hardly seemed worth while.

The younger the patient at the onset of symptoms the worse the results. An individual who breaks down in his thirties, expecially after some precipitating cause has a much better chance than one who breaks in his teens. The latter can frequently return to his job or resume housekeeping. Prognosis is worse in children.

It comes as somewhat of a surprise to find the hebephrenic schizophrenic responds just as favorably, if not more so, than the paranoid and catatonic. The probable explanation is that he is less aggressive and the convalescent care is easier to carry out; he is more likely to carry out suggestions and commands of his family or employer.

The operative mortality is 2 per cent. Twelve per cent of the patients develop convulsive attacks. If the patient's condition is not considered serious enough to warrant taking these risks, prefrontal lobotomy is not indicated.

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CONGENITAL ANOMALIES OF THE NEURAL AXIS*

SURGICAL MANAGEMENT BASED ON EMBRYOLOGIC CONSIDERATIONS

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PLANS for surgical intervention in a patient with a congenital malformation at any point along the neural axis should be directed toward improving the chances for normal future development and prevention of further deformity. When patients are born with visible malformations such as spina bifida or cranium bifidum, neurologic deficit may be apparent at once or may develop only after a period of growth. Patients in both categories may have their condition ameliorated or cured by judicious surgical intervention or orthopedic care. It follows that the ability to make an estimate of the embryologic deficit of a patient is as important as gauging the degree of the neurologic deficit. Growth not only may augment the deformity but may also render irreversible changes which were once reversible. Therefore, judgment in the selection of individuals for surgery, as well as the time and type of operation, should be based upon embryologic as well as neurologic knowledge. One should not attempt to correct an insurmountable anomaly only to leave a child unable to become physically and economically independent.

Existing knowledge of human embryology is almost wholly limited to morphologic description of the normal and the abnormal. Because great technical difficulties hamper experimentation in the mammalian field, the most extensive experimental advances have come from work on amphibians and the chick. Conclusions based on investigation in one species can be applied to different species only with reservations. Nevertheless, the recapitulation of phylog-

eny by ontogeny permits conjecture that some of the basic mechanisms of embryology in lower forms must carry over, at least in principle, in man. Consequently, a brief discussion of the factors controlling embryonic development is in order.

The blastopore of an early embryo is a collection of totipotential cells. The three primitive germ layers, ectoderm, mesoderm and endoderm which arise from this center of growth extend cephalad in contrast to the primitive streak which lies caudad. Formative movements, such as invagination, lateral expansion, longitudinal extension and dorsal convergence, so shape the interstitial growth of tissues derived from the primitive germ layers, that further morphogenesis leads to completion of the embryonic system. Spemann[1] questions whether the faculty of formative movement in embryonic tissue may not be determined as early as fertilization in vertebrates. He later suggests that harmony of decisive extrinsic factors is necessary to stimulate integrated movements which are intrinsic qualities of competent tissue. In embryologic parlance, this process is termed induction. Needham[2] considers tissue to be competent when it is able to react to a given morphogenetic stimulus in such manner that determination of histogenesis will lead to proper organogenesis. Workers in Spemann's laboratory[3] have achieved induction of neural tissue from undetermined ectoderm of amphibia by the application of a chemical extract of tissue excised from the region of the blastopore of other embryos. This leads Spemann to conclude "that inductive action is trans-

ferred by a chemical substance." In the embryologic literature such chemical substances are referred to as *organizer substances*. An integrated and synchronized system of induction appears to reign during the elaboration of the embryo, with each system and its integral parts adhering to a strict schedule. From experimental evidence, Spemann[4] points out that tissues from which the neural structures arise are not susceptible of induction until after the mesodermal substratum has formed, and then for only a brief period. Gilchrist[5] by an ingenious method of directing heat to any given point on the neural axis of incubating newt's eggs, demonstrates that there are temporal limits for determination of individual elements of the neural axis. He further determined that a thermal disturbance applied within such temporal limits so alters the physiology of the part affected that the local pattern of determination will be abnormal.

If the embryo is inspected at the stage of invagination of the blastopore (gastrulation), differentiation of the three primary germ layers is apparent as the preface to future organogensis. Interruption at the next stage, neurulation, reveals elaboration of the neural axis. At this time, the general pattern of the embryo may be foreseen in the concomitant somite formation. A slow-motion viewing of this process reveals that the ectoderm from which the central nervous system is derived appears first as a groove between two rising crests. Needham[6] points out from avian studies that the blastopore moves backward, followed by the folds of the neural crests, "as if it were drawing the latter as a pencil would do." Bremer,[7] on the other hand, believes that the neural axis grows primarily forward from the neuropore, thus causing the projection of the head. The folds converge dorsally to form the neural tube by fusion. In man, fusion of the neural tube is initiated between the twenty-first and twenty-second day after ovulation. Bremer[8] considers this initial point of fusion to be the site of the caudal end of the hindbrain.

Normally, fusion is completed at the cephalic pole of the neural axis between the twenty-fifth and twenty-sixth day and caudally by the twenty-ninth day. As closure of the neural tube progresses, concomitant encasement with mesenchyme causes it to appear to sink beneath the surface ectoderm. Mesenchyme derived from the mesoderm of the supporting somites, in addition to supporting and insulating the neural tube, has the prime function of inducing it. It is at this crucial embryonic stage,[8] the twenty-first to twenty-ninth day after ovulation, that the groundwork is laid down for malformations, such as spina bifida and cranium bifidum with or without meningeal protrusion. Myelomeningoceles and encephaloceles accompanying spina bifida and cranium bifidum may be construed as arising from faulty induction of ectoderm, secondary to the mesenchymal defects. Patten[9] reports two human embryos, aged seven and six weeks, respectively, with malformations suggestive of encephalocele and myelomeningocele formation. He believes that lateral overgrowth of neural tissue preventing dorsal convergence is the cause of failure of closure of the neural tube in these cases.

For purposes of discussion, it is preferable to look upon the conditions found in Patten's two embryos, and all neural malformations along the neural axis, as abnormalities of neurulation. Bifid conditions, cranial or spinal, with or without meningeal protrusion in conjunction with abnormal overgrowth of superficial blood vessels or fat may be considered the result of faulty mesenchymal induction. Gastrulation and neurulation depend upon the essential morphogenetic movements termed invagination and dorsal convergence, respectively. Exogastrulation in amphibia, through reversal of invagination, has been produced at will by Holtfreter,[10] and Curtis et al.[11] The former achieved this reversal of formative movement by maintenance of amphibian blastulae in 0.35 per cent saline solution, while the latter achieved similar results by irradiation. These environmental

abnormalities apparently interfered with the reaction of organizer substances and competent tissue in such a manner as to reverse the direction of morphogenetic movement.

Recently, Ancel[12] has observed myelomeningocele formation following inoculation of fertilized chick eggs with various chemical agents between the sixteenth and twenty-sixth hours of incubation. He has achieved the same result by incubating the eggs at 40.5°C. instead of 37°C. His experiments reveal that the earlier chemical or physical disturbances take place, the higher the deformity will lie on the neural axis. He concludes that myelomeningocele formation is dependent upon faulty mesenchymal induction.

Snell, Bodeman and Hollander[13] have produced gross abnormalities of the central nervous system, similar to that described by Patten[9] as the precursor of an encephalocele by irradiation of male mice prior to mating with normal females. In this instance, it might be construed that the genetic change which followed irradiation had rendered the tissue incompetent to respond in a normal manner to the morphogenetic stimuli or the organizer substances incapable of evoking appropriate stimuli.

In man, the cause of spina bifida, cranium bifidum, meningeal and neural malformations is not known. The experimental work on amphibia, birds and lower mammals mentioned above suggests that possibly, in man, similar aberrant factors may adversely influence neurulation. Thus, abnormalities of neurulation may result from incompetence of mesoderm and ectoderm, or the reaction of organizer substances so altered in quality or time or action, that the resulting stimulus leads to abnormal induction. The extent of the anomaly will depend upon the degree and duration of imbalance and the area in which it operates. Therefore, the protean forms of spina bifida, cranium bifidum with vascular, fatty and meningeal defects as they are seen clinically, may be quite reasonably postulated in view of the multipotential

nature of mesenchyme. It should be remembered that mesenchyme is a primitive tissue from which corium, fat, fascia, muscle, blood vessels, the components of the vertebral column, notochord, chondral and membranous portions of the skull and some portions of the meninges are differentiated but not the neural elements.

It is now appropriate to examine the embryologic evidence which will throw light upon the anomalies, ranging from the minor myelomeningocele and encephalocele, through anencephaly. In these anomalies there is neuroectodermal malformation in addition to the mesenchymal defect.

Exogastrulation studies such as Holtfreter's[10] have demonstrated that with complete evagination of the blastopore (exogastrulation) no neural tube differentiation is possible. Needham,[14] in discussing exogastrulation, makes the statement that, "The underlying of the dorsal ectoderm by mesoderm is the full, perfect, and sufficient cause for its determination for neural differentiation."

Holtfreter's[15] later work with explanation of not fully determined tissues further confirms Needham's hypothesis. Holtfreter discovered, as is illustrated in Figures 1 through 5 that proper neural induction is dependent upon normal development of mesoderm.

Conditions depicted in Figures 2 and 4 are suggestive of hydromyelia in man; likewise Figure 3 is suggestive of myleomeningocele formation as seen in man. Possibly the factors producing these abnormal inductions under experimental conditions may exist in the form of mesodermal deficiencies in the human embryo when faulty neural induction leads to hydromyelia and myelomeningocele. In other words, clinical lesions ranging from a minor neural malformation associated with bifidism of the neural axis to complete rachischisis of the spinal column with dysraphism, or anencephaly, will depend upon the degree in which the mesenchyma is defective and, therefore incapable of proper neural induction.

A detailed discussion of the origin of teratoma and teratoid tumors is beyond the scope of this paper because of their neoplastic nature. However, because of their frequent association with congenital anomalies of the neural axis, they deserve mention here. The differences in life history and histological nature of the teratomas arising in the sacrococcygeal region as compared with those found elsewhere along the neural axis, makes it feasible to consider them separately.

The sacrococcygeal group of tumors are truly teratomatous in nature, constant of position and massive in growth with a high

state of cellular differentiation at the time of birth. These characteristics suggest that they may result from tissue incompetence, or disturbance of organizer substances occurring at the blastopore when induction of the neural axis is being completed.
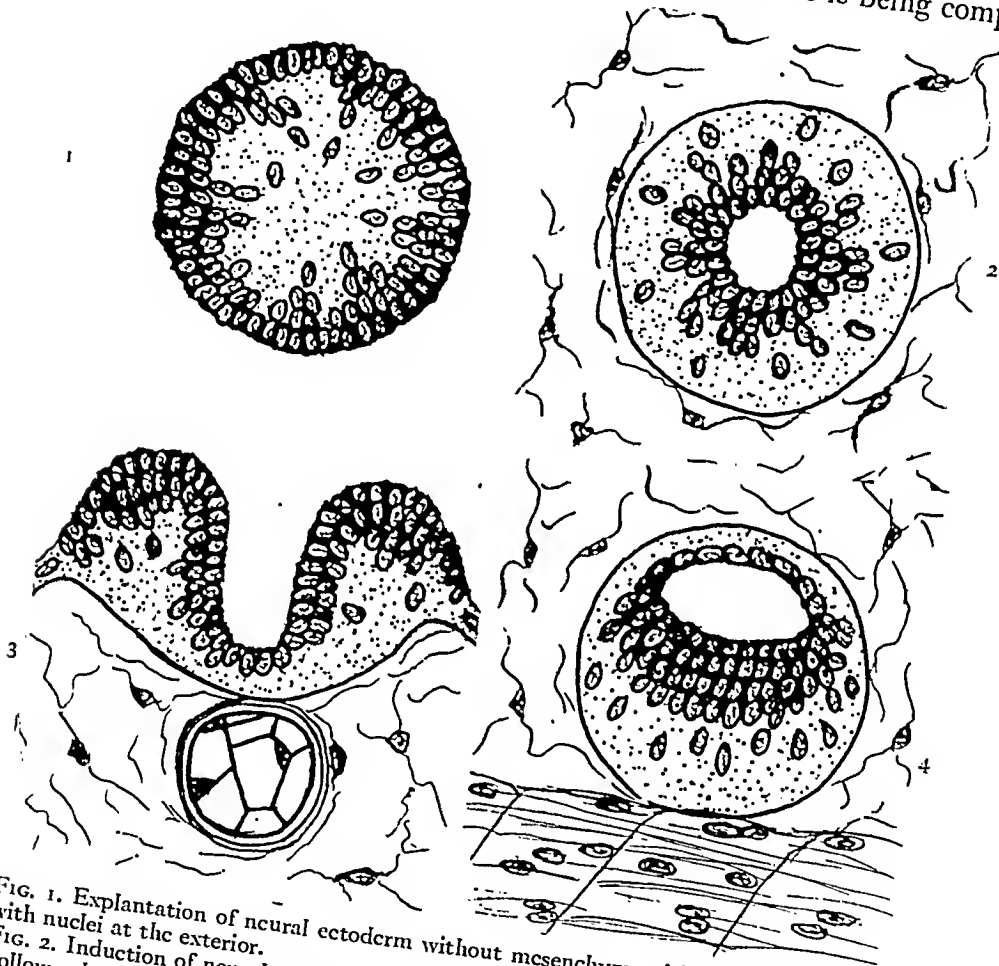
Fig. 1. Explantation of neural ectoderm without mesenchyme yields a sphere or rod with nuclei at the exterior.
Fig. 2. Induction of neural ectoderm within mesenchyme without notochord yields a hollow sphere or rod with nuclei centrally located.
Fig. 3. Induction in the presence of notochord with insufficient mesenchyme yields a failure of closure of the neural tube with cell bodies lying along the free surface and thinning of the neural plate in contact with the notochord.
Fig. 4. Induction on muscle yields a tube with eccentric lumen.

Holtfreter's[10] work with amphibia suggests that exogastrulation, causing dislocation of essential toti- and multipotential cells, plays an important part in teratoma and teratoid formations along the neural axis. Exogastrulated amphibia developed teratoid growths in his experiments at the time that neurulation should have been taking place. Ingraham and O. T. Bailey's[16]

study of teratoma and teratoid tumors in man reveal evidence of congenital anomaly in several cases. This might be attributed to faulty neurulation following a disturbance in gastrulation. Further speculation may be considered futile in view of ignorance of the cause of neoplastic formation.

Persistence of remnants of the neurenteric canal is an extremely rare anomaly. Bremer,[17] in a review of the literature, found but three cases and attributes them to persistence of accessory neurenteric canals. Since the cases reported in the literature and the ones studied in this clinic have all been associated with spina bifida, it seems appropriate to introduce them in this discussion and to consider the possibility that they may result from an aberrant morphogenetic movement occurring late in gastrulation or early in neurulation.

Avian and human individuals both show incidental occurrences of spina bifida. Ancel's[12] studies reveal a 0.25 per cent incidence of this condition in normal control chick embryos. Ingraham and Lowrey[18] report from this clinic that approximately 25 per cent of normal children have bifid spinal conditions which are incidental findings in x-ray films taken for some other reason. Whether these variations from the normal are the result of adversive genetic action or alterations in the environment of the hen's egg or the human uterus, is a question that is far from settled. Murphy,[19] after extensive statistical and environmental study of families possessing malformed children, comes to the general conclusion that, "Gross human congenital malformations arise solely from influences which affect the germ cells prior to fertilization." In the course of his research, he was able to demonstrate that a congenital malformation will occur with twenty-five times greater frequency than that of the general population when an offspring is born to parents already possessing a malformed child. The more recent report of Gregg[20] tends to offset the pessimism of Murphy somewhat. The latter author shows an association between development of rubella

in the mother and the occurrence of congenital ophthalmologic and cardiac defects in the child. This leads him to the speculation as to whether the cells most actively engaged in crucial points of organogenesis may not be so affected by the disease proc-
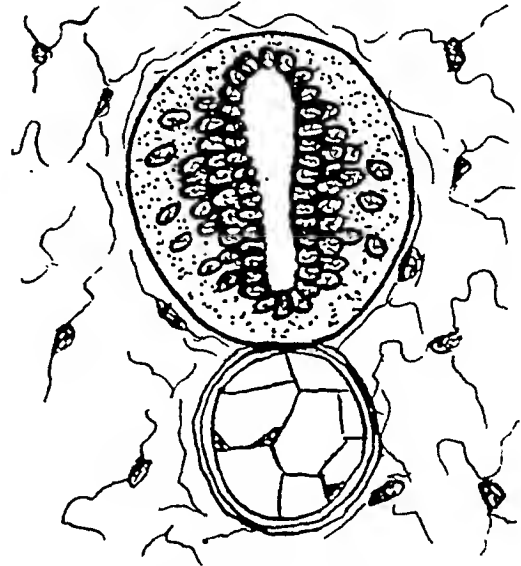
Fig. 5. Induction in the presence of sufficient mesenchyme and in contact with notochord yields a neural tube of proper contour.

ess that aberrant development results. Ingraham and Swan[21] report from this clinic in a study of 277 patients with spina bifida, that the family histories revealed a 6 per cent incidence of spina bifida and approximately the same incidence for other congenital anomalies. It is interesting to note that more recently in this clinic a few cases with major neural axial deformity have been recorded with a negative family history for congenital deformity, whose mothers and attending physicians can substantiate the fact that an acute upper respiratory infection with associated hyperthermia to 104–105°F. developed during the third and fourth week of gestation. In light of present knowledge it is doubtful whether the human intrauterine environment can be influenced by factors other than temperature without having the embryo so altered that it becomes spontaneously aborted. The research of both Gilchrist[5] and Ancel,[12] on lower invertebrates, which shows that the morphogenesis

of the neural axis may be affected by hyperthermia presents an interesting correlation with Gregg's[20] clinical observations. Needless to say, many more cases of proven hyperthermia at the time of neurulation, similar to the few recently studied in this clinic, will have to be accumulated before the pessimism of Murphy's conclusion can be lifted. However, it is to be hoped that other members of the profession will take an interest in this subject in order that a true evaluation of the causative factors of congenital anomalies, genetic and environmental, may be obtained. Undoubtedly, certain matings are genetically doomed to produce congenital anomalies of the neural axis, but it is possible that the occurrence of a certain percentage is due to a fortuitous circumstance such a hyperthermia coincident with neurulation. When this tremendous problem has been solved, doctors may, with more assurance, advise parents concerning the desirability of having additional children after the birth of a deformed offspring.

SURGICAL CONSIDERATIONS

Bearing in mind the foregoing comment on basic embryologic factors that may form the background for the origin of congenital malformations, attention will be turned to the surgical aspects of this discussion. Tempering of neurologic judgment with an embryologic concept will aid the surgeon in the selection of cases for operation as well as the time for and type of operation. Embryologic data pertinent to the anomaly under consideration will be presented wherever feasible. By careful individual evaluation, Ingraham and Swan[21] show in a series of 546 cases with spina bifida and cranium bifidum that 30 per cent of the former and 34 per cent of the latter may expect a relatively normal existence when aided by appropriate surgical measures. These figures tend to allay the pessimism of the past concerning the prognosis of patients born with neural axial deformities.

GENERAL ANATOMIC CONSIDERATIONS OF SPINA BIFIDA

The supporting structures of the neural axis are differentiated from mesenchyme which is, itself, mesodermal in origin. If the mesenchyme which develops parallel to the neural crests fails to encase completely the neural tube by dorsal or ventral fusion, spina bifida occurs. Dorsal spina bifida is much more common than the ventral type. Meningeal protrusion through the bifid defect constitutes a meningocele. This phenomenon probably occurs when all encasing mesenchymal elements, except the meninges, fail to fuse. Myelomeningoceles in man arise in association with spina bifida. Ancel[12] concluded from his experimental work that myelomeningocele occurs in the chick in conjunction with spina bifida as the result of faulty induction of neuroectoderm by mesoderm which in itself is so inadequate as to lead to spina bifida. That this may possibly be the case in man is not hard to conjecture.

The results of Holtfreter's[15] explantation experiments, in which he encased medullary plate of amphibians with mesenchyme, point out that neural induction in amphibia depends upon a mesodermal factor. Certainly the configuration of some of his abnormal inductions achieved by varying the amounts and components of mesenchyme, are suggestive of hydromyelia (Fig. 6) and myelomeningocele formation seen in man. Diplomyelia (Fig. 7), which occurs occasionally in patients with myelomeningocele, is thought to be a manifestation of partial twinning by Herren and Edwards.[22] Bremer,[8] in discussing gastrulation in amphioxus, the lowest form of chordate, points out that the nervous tissue lying in either lip of the gastrula mouth, later known as the neurenteric canal, becomes the homologue of the paired neural plates from which the central nervous system is developed. He later states that in higher vertebrates such as amphibia, gastrulation is passed through so rapidly that the paired neural plates on either side
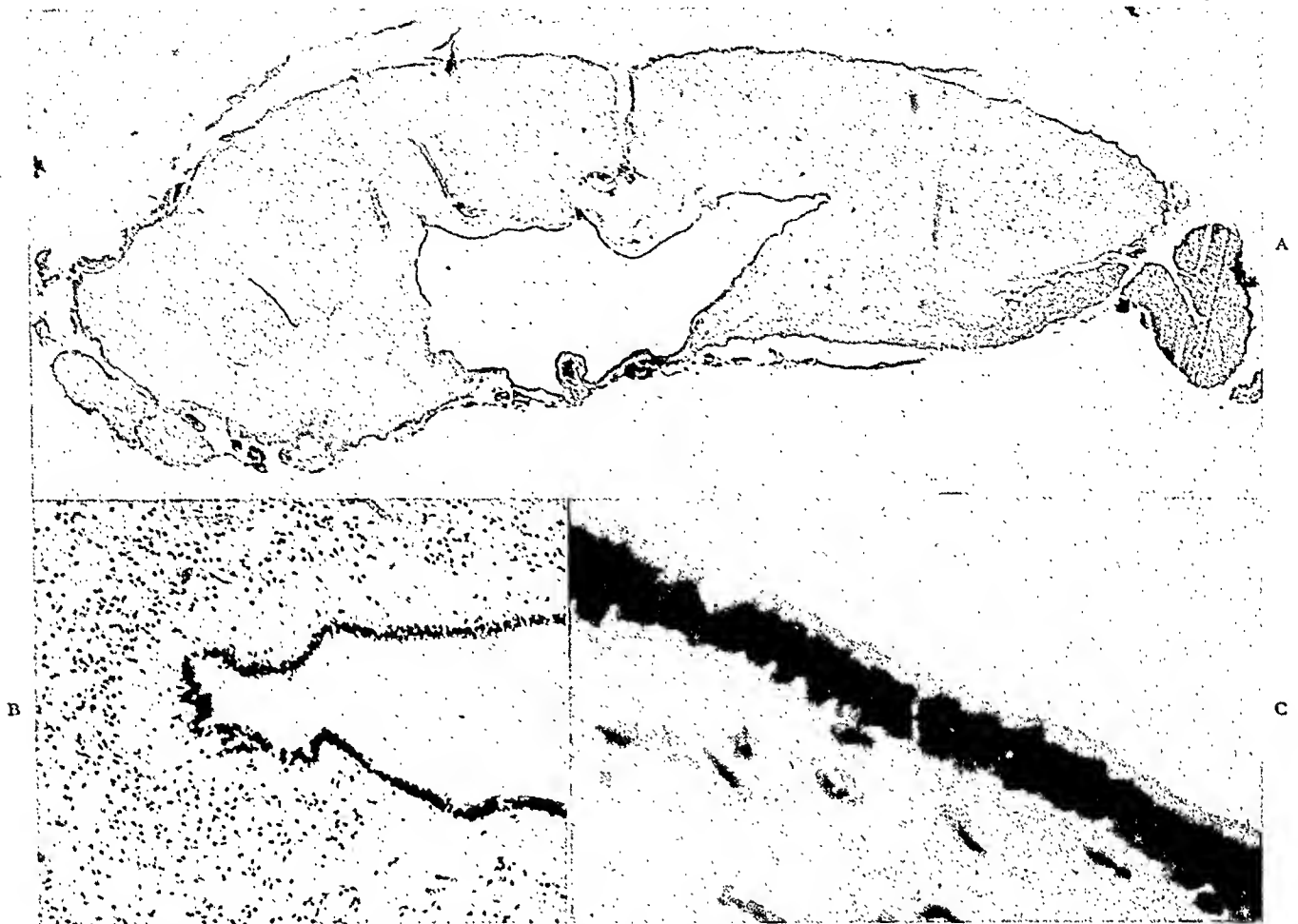
FIG. 6. A, photomicrograph of a high thoracic section of spinal cord showing hydromyelia. Hematoxylin and eosin stain. Magnification 15 diameters. B, magnification 130 diameters to show ependymal lining of the cavity. C, magnification 500 diameters to show further detail. This patient had a successful resection and repair of a thoracic myelomeningocele at the age of eleven months. Death resulted from an intercurrent gastrointestinal infection five weeks after operation.
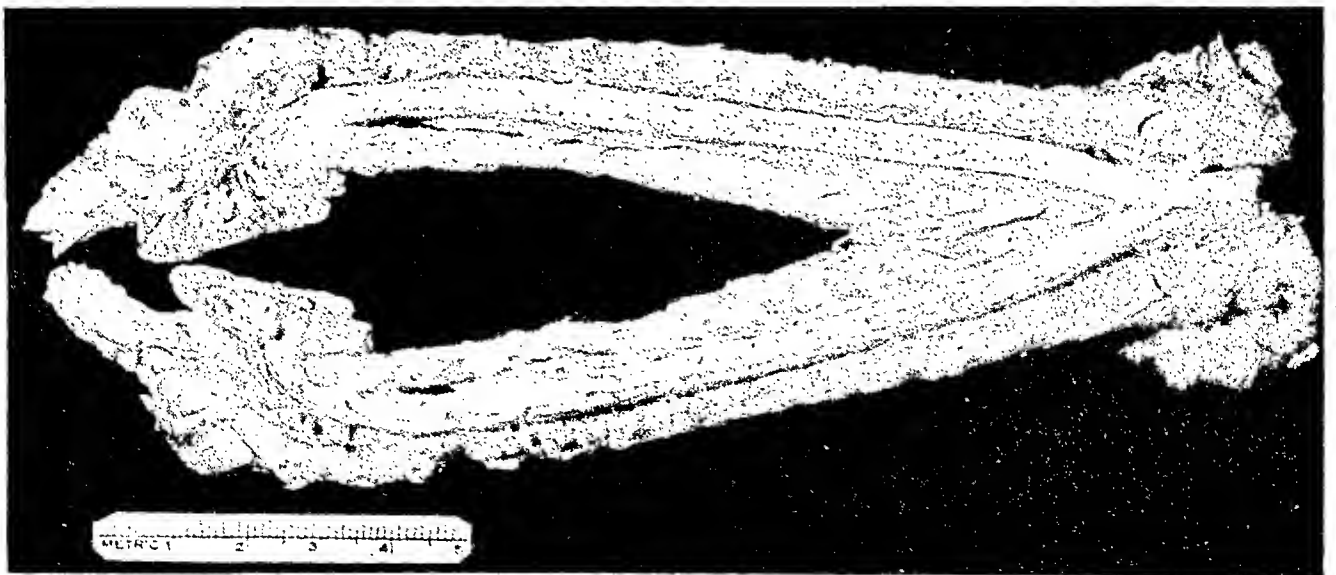
FIG. 7. This photograph of an autopsy specimen shows diplomyelia. The duplicated spinal cord ends in a lumbo-sacral myelomeningocele.

FIG. 8. This photograph reveals' hypertrichosis over the site of a spina bifida occulta in a girl aged five years.

FIG. 9. This photograph reveals a dermal sinus situated in the upper sacral region over a spina bifida occulta. The dermal sinus which led down to an intrathecal teratoid tumor was surrounded by telangiectases.

of the neurenteric canal giving rise to the nervous system, appear only as a fused neural groove growing forward from the blastopore. This author mentions experimental work in which the ovum is so damaged that the gastrula lips pursing the neurenteric canal do not fuse. Such situation, he points out, leads to diplomyelia in lower forms. With this experimental evidence at hand, it may be possible to imagine that diplomyelia in man may arise as a result of some morphogenetic disturbance in the stage of gastrulation.

Mesenchyme contains the mosaic for the fat, blood vessels, fascia, ligaments, bones, corium and some elements of the meninges. It is, therefore, not surprising that when spina bifida occurs, both fatty and vascular growths as well as aberrant fibrous strands should develop concomitantly with the bony defect. Abnormal fat overgrowths developing simultaneously with meningoceles and myelomeningoceles are so common that Ingraham and Swan[21] refer to

these conditions as lipomeningocele and lipomyelomeningocele. Spina bifida occulta is a common defect in the general public. Ingraham and Lowrey[18] report a 25 per cent incidence in normal children. This condition obtains clinical significance only when neural elements are impinged upon by aberrant bone formation, fatty and vascular masses, or when constricted by adhesions. Surface manifestations of spina bifida occulta include hypertrichosis (Fig. 8), lipoma and angioma formation, dimpling of the skin, dermal sinuses (Fig. 9) or bony protrusions.

SURGICAL INTERVENTION IN SPINA BIFIDA OCCULTA

Bremer,[23] quoting his teacher, C. S. Minot, states that from a relative point of view, 90 per cent of growth and development is accomplished *in utero* when one considers what transpires between zygote formation and term as compared with term and full maturity. It follows then that surgery has little to offer the patient born with a major deformity and marked neurologic deficit, prenatal in origin. Such lesions may be looked upon as "fixed." On the other hand, patients born with a minor deformity who show increasing neurologic deficit as a result of interference with the dynamics of

ascent of the spinal cord after birth may be considered to be in a plastic state. The possibility of amelioration by surgery is far greater in this group provided it is carried on while the physiologic changes are still reversible.

At this juncture an account of the mechanism of ascent of the cord will be useful in illustrating why some neurogenic deformities occur and in selecting individuals for treatment. The caudad formation of the neural axis during the second month of gestation progresses so far that the blastopore finds itself at the tip of a free tail. This structure likened to a tail is a coccygeal continuation of the spine, complete with hind gut, containing spinal cord supported by somites and notochord. The primitive streak, in the meantime, has sought the ventral surface of the embryo from the tip of the tail to the body stalk. Toward the end of the second month, fusion of the vertebral elements of the tail occurs with concomitant ventral retraction within the soft tissues to form the adult coccyx. At this time, by virtue of vesiculation and degeneration, all coccygeal neural elements up to the first coccygeal nerve so deteriorate as to be recognizable only as the filum terminale. The hindgut likewise becomes fragmented and is absorbed. From the third month on, the spinal column grows more rapidly than the cord. This discrepancy in rate of growth normally leads to ascent of the cord through traction exerted by the relative fixation of the structures of the hindbrain within the calvarium. Under ordinary circumstances, at term, the tip of the conus medullaris arrives at the third lumbar level through constant traction. Therefore, the degree of faulty mesenchymal induction will determine the range and development between an inconsequential bifid defect and a massive one with aplasia and dysplasia of many of the supporting elements of the vertebral column. Varying degrees of neurologic deficit will occur in direct proportion to the distortion of the cord by adhesions, osseous and fatty masses. Acceleration of the rate of growth

will increase the deficit because of the unbalanced traction exerted by ascent of the cord. It follows then that a severe degree of neurologic deficit may exist at term. Thus, neurologic deficit may never exist, or may vary from a minor sensory, motor or trophic change to a para- or quadriplegia with sphincter incontinence. Some of the commonest signs are underdevelopment of a foot or extremity, maldevelopment such as a valgus, varus or cavus deformity of a foot or weakness in a major muscle group of a lower extremity. The commonest symptoms are disturbances of gait, station or posture. Trophic disturbances with signs of sensory defect are seen as well. Failure to respond to bowel training or loss of previously established bowel and urinary control points to involvement of sphincters.

In infancy and childhood many valuable signs can be detected by observation of the patient at play or in moments of struggle. In addition, a detailed orthopedic and neurologic examination is possible with but few limitations because of age. Reflex changes can be elicited with reliability. As far as sensory deficit is concerned, failure of withdrawal and lack of vocal or lacrimal response to painful stimulation is reliable in the very young. Distraction with a nursing bottle or toy is useful as an adjunct to this examination in order that the patient may become quiet between stimuli. It is the experience of this clinic that no patient is too young for evaluation.

X-ray examination of the spine and extremities is useful in determining what particular bony abnormality is present. (Fig. 10.) The limitation of interpretation of spinal x-ray studies, however, is in direct proportion to the amount of calcium laid down in the chondral scaffold of the age group in question. Air and pantopaque myelography is a useful adjunct when the presence of an intraspinal defect or mass is in question. (Fig. 10.) Air is used in the younger age group to avoid the necessity of withdrawal of the contrast medium. In older infants, 1 to 3 cc. of pantopaque is recommended. A general anesthetic is
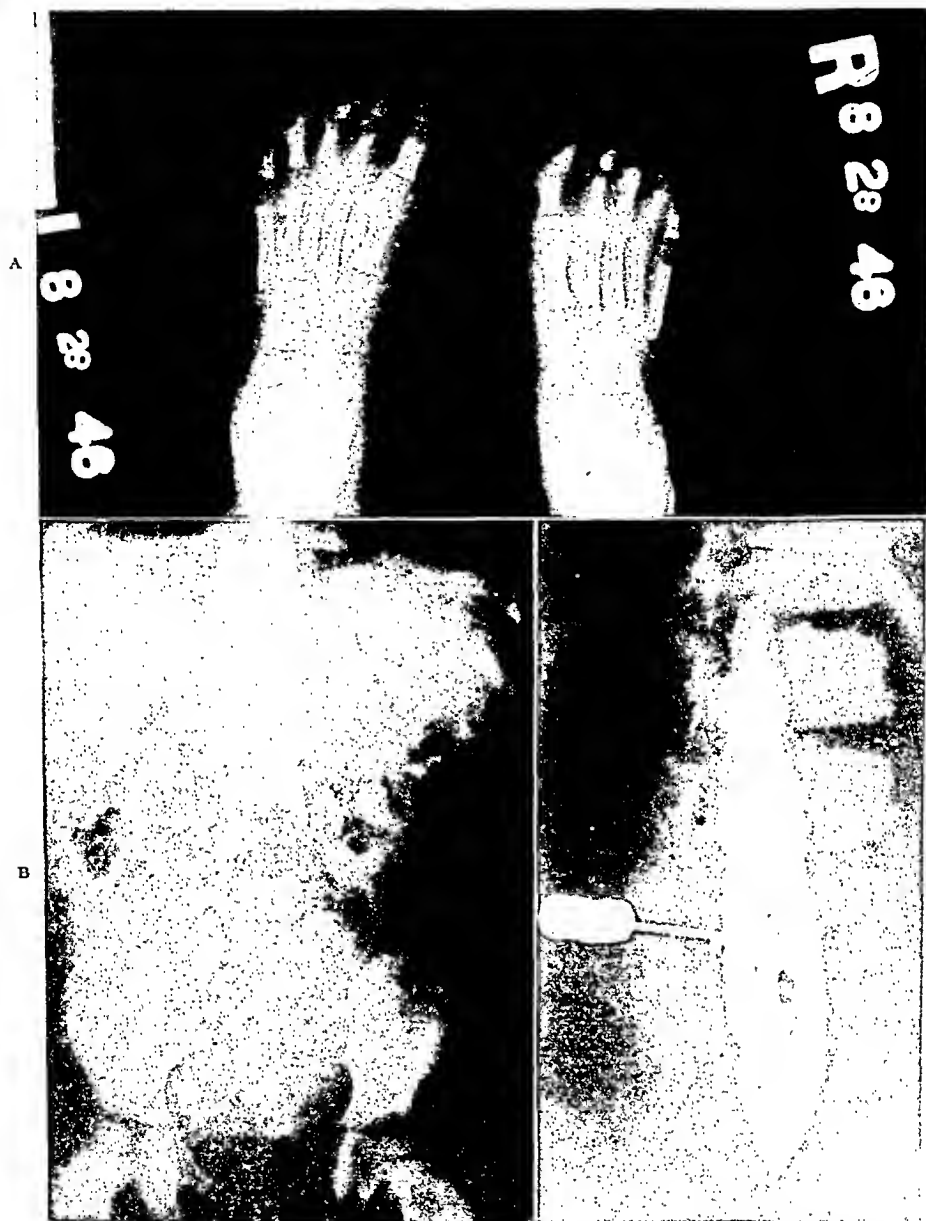
FIG. 10. A, photograph of roentgenogram of the feet of a patient aged one and one-half years born with a lumbar, sacral and coccygeal spina bifida occulta. Note the underdevelopment of the right foot as compared with the left. B, photograph of roentgenogram revealing the spina bifida occulta. C, pantopaque myelogram revealing abnormal bony spinule situated in the midline at L3. At operation this was found to be adherent to and impinging upon the right half of the cauda equina.

necessary to permit safe handling of the column of this contrast medium and to avoid spillage into the basal cistern and intracranial subarachnoid spaces. Introduction and withdrawal of the contrast medium is handled in the manner described by Kubik and Hampton[24] Urethrography, cystography and cystometry are of aid in distinguishing genitourinary tract anomalies from bladder disturbances of neurogenic nature.

PREOPERATIVE PREPARATION

In general, preoperative preparation of patients with spina bifida and cranium bifidum follows the principles outlined below. Fluids are restricted for at least six hours before operation; enemas are given
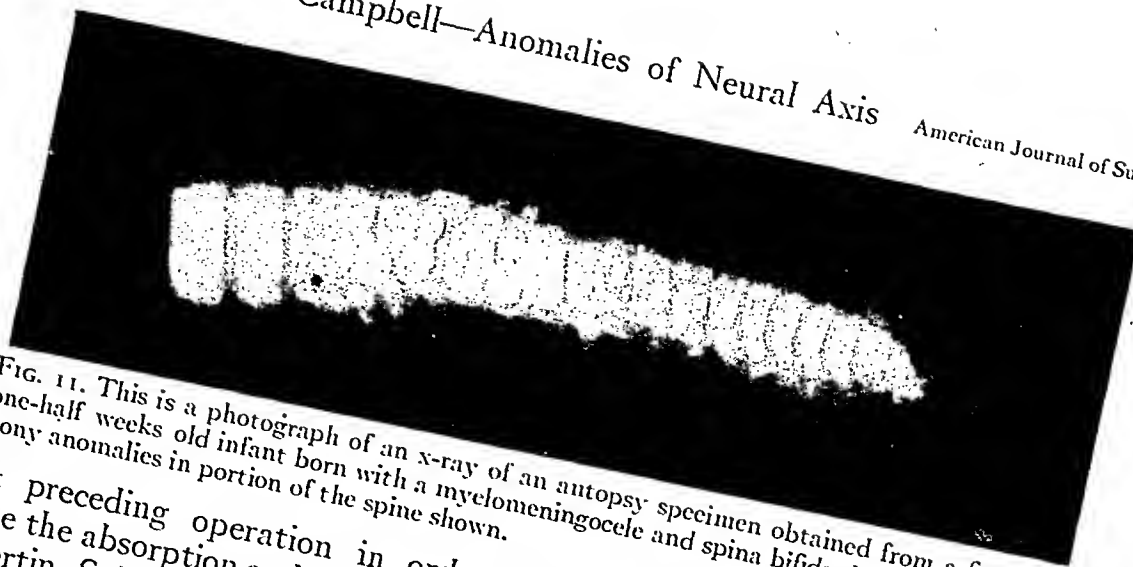
FIG. 11. This is a photograph of an x-ray of an autopsy specimen obtained from a five and one-half weeks old infant born with a myelomeningocele and spina bifida. Note the multiple bony anomalies in portion of the spine shown.

the night preceding operation in order to facilitate the absorption and administration of avertin. Sulfadiazine and penicillin are administered prophylactically when the operative site lies in the lumbar region or below, or when the crevassing or the granulations on the surface of the meningocele will not permit confidence of sterilization of the operative field with the usual chemical agents. Great care must be given to walling off the rectum from the operative field when a low exploration is planned. This is carried out before the skin preparation starts. No major procedure is started without the insertion of a constant intravenous cannula.

ANESTHESIA

In this clinic, most operations are carried out under avertin-ether anesthesia, supplemented by local infiltration of 0.5 per cent procaine with adrenalin 1:1000 added to the amount of 0.3 cc. to each 30 cc. Avertin is administered to children in the dose of 100 mg. per Kg. of body weight, and 80 mg. per Kg. of body weight in infants. Avertin is not administered to the newborn or to patients suffering from cachexia, renal or hepatic disease or to patients upon whom a low spinal exploration is contemplated for fear of contaminating the incision during operation. Intratracheal ether is administered with cervical or intracranial lesions in which embarrassment of the respiratory center during operation is likely. Anesthesia in the newborn is given in the form of open drop ether or brandy 4 to 5 cc. by stomach tube in addition to local procaine infiltration. When the infant awakens during the procedure, a nipple stuffed with cotton saturated with brandy diluted 1:8 is offered. This usually leads to further relaxation through raising the blood alcohol level.

SURGICAL TREATMENT OF SPINA BIFIDA OCCULTA

A midline incision is made from the first spinous process above and below the bifid area, after having been so draped that extension cephalad and caudad is facilitated if indicated. The laminae of the first normal superior and inferior vertebrae should be exposed before carrying the incision beyond the superficial fascia in the anomalous area. Great care must be exercised to prevent entering the dura or possibly the cord during dissection. Every adhesive band, until proved otherwise, should be looked upon as a peripheral nerve root. Findings at operation vary tremendously. Anomalous osseous arrangements involving the spinous processes, laminae, pedicles, facets and vertebral bodies are encountered in any combination. (Fig. 11.) Five cases of aberrant midline spicules of bone attached to the dorsal surface of a vertebral body impinging upon nerve roots, are on record in this clinic. Reversal of clinical signs in "non-fixed" lesions can be obtained by careful and judicious removal of these bony malformations with due regard being given to avoiding root evulsion and laceration of the cord. Fatty malformation, subcutaneous, extradural and intradural, is frequently encountered in the surgery of spina

A   B

FIG. 12. A and B; these photographs were taken when the patient was two years old during the operative removal of an intrathecal lipoma shown in C.

FIG. 12. C, The lipoma was lumbosacral in position. The presenting symptom was poor rectal sphincter control. There were no neurologic signs except a questionably weak rectal sphincter. An upper sacral subcutaneous lipoma overlay a spina bifida occulta. 3×.

bifida occulta. A discrete tumor can be removed with reversal of signs caused by its impingement, provided intervention is timely. (Fig. 12.) Unfortunately, all lipomatous formations are not discrete. However, decompression afforded by exploratory laminectomy will relieve pressure and alleviate signs resulting from the extradural type. Likewise, decompression benefits the patient with diffuse lipomatous infiltration of the cauda equina and cord itself. Fibrous tracts, possibly sclerosed meningoceles or dermal sinuses lead down through the dura to the cord. Lysis of adhesions associated with the latter type of malformation, as well as with the lipomatous and osseous anomalies, may afford the greatest benefit because of the relief from the distortion of unbalanced traction placed on the growing spinal cord and nerve roots.

SURGERY OF MENINGOCELE AND MYELOMENINGOCELE

The clinical distinction between a meningocele and a myelomeningocele is most difficult and is more accurately made during operation or when pathologic studies are complete. Ingraham and Swan[21] adopted the following clinical criteria: "If a sac exists clinically and nerve elements cannot be seen, and if there is no evidence of neurologic disability as demonstrated by muscular weakness or paresis, loss of sphincter tone, or aberration in cutaneous sensation, the lesion is a meningocele. (Fig.

13.) If nerve elements are visible or neurologic disability exists, the lesion is a myelomeningocele. (Fig. 14.)" As has been shown before, meningocele and myelomeningocele formation is contingent upon a bifid condition of the spine. The former probably arises as the result of improper induction of all mesenchymal elements down to those containing the mosaic for the meninges: the latter may be attributed to abnormalities of neurulation involving the ectoderm as well. Abnormalities of neurulation of greater severity may account for peripheral nerve aplasia due to neural crest malinduction, hydromyelia or diplomyelia.

Therefore, in selection of patients for surgery and determination of the appropriate time for operation, the prevention of meningitis must be considered as well as the criteria used in cases of occult spina bifida. At the same time, the over-all prognosis is poorer when a myelomeningocele exists. This implies the addition of ectodermal malformation to the mesenchymal one of the accompanying spina bifida. Position, size, contour, condition of the herniated meninges and availability of tissue for closure are all factors that must be evaluated before attempting operation upon a meningocele. Thus, thin, pedunculated meningoceles with small stalks should be submitted to surgery as early in the neonatal period as the condition of the patient will permit, provided the patient does not in another way appear to be defective. Rupture of the sac, during delivery or soon afterward, is an indication for immediate intervention, as is incipient ulceration. In such cases, prompt surgical attention, combined with chemotherapy, has yielded gratifying results. Unfortunately, the broad based sessile type of meningocele is more common than the pedunculated. Inadequate tissue for closure of this type of defect, or frank ulceration precludes surgery in spite of the risk of meningitis from rupture or further ulceration of the membranous covering. Under these circumstances, protective dressings should be ordered. These consist of a layer of sterile perforated cilkloid over

the surface of the meningocele and an outer layer of gauze. Around this a protective ring of sheet wadding, wrapped in gauze, is maintained in position by a binder. Epidermis propagates beneath the perforated cilkloid to yield the necessary protective barrier. Daily, and more often if indicated, the dressing is taken down to the cilkloid
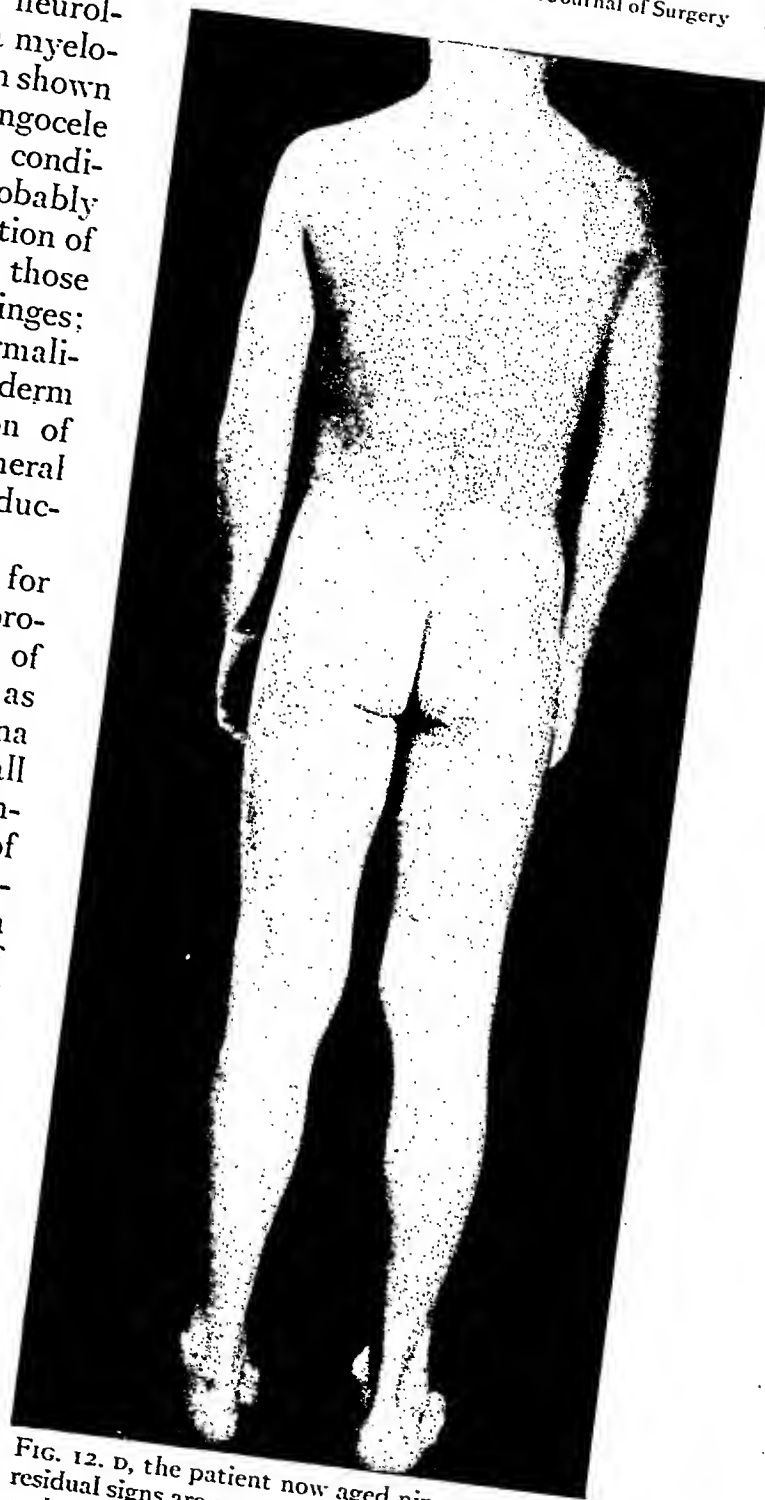
FIG. 12. D, the patient now aged nine years. The residual signs are occasional urinary incontinence and slight motor and sensory deficit in the lower extremities.

and cleansed with boric acid solution. The cilkloid is replaced as necessary, but not too frequently because of the deterrent effect of frequent changes upon epithelialization. When epithelialization is complete and adequate tissue for closure has de-

child provides a stronger patient and lessens the operative risk. Operation is facilitated by more adequate tissue for repair and closure. In addition, intrinsic defects not detectable in the newborn come to light during these months of observa-

FIG. 13. Diagrammatic drawing of a meningocele. A, saggital section. B, coronal section.
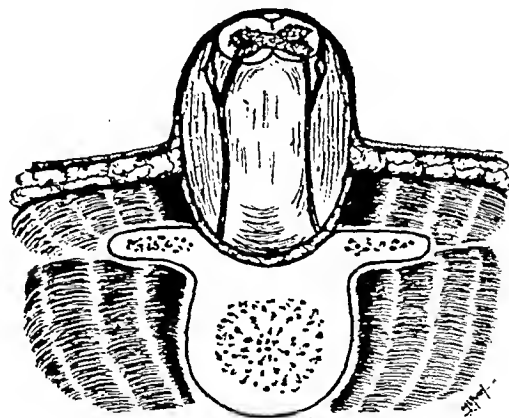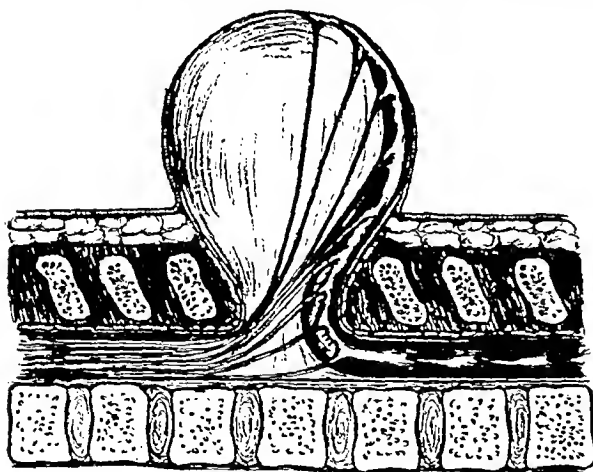
FIG. 14. Diagrammatic drawing of a myelomeningocele. A, saggital section. B, coronal section.

veloped, operation is considered. In the event of meningitis, surgery is deferred in favor of polyvalent chemotherapy which is instituted after taking cultures. The age of choice for operation is twelve to eighteen months unless circumstances force earlier intervention, at a time when growth of the

tion. Hydrocephalus or other outstanding deformities contraindicate operation. (Fig. 15.) A certain number of patients thought to be improper subjects for surgery as the result of hydrocephalus, neurologic deformity or questionable mental status may compensate in a two-year period.

Therefore, it is a wise policy to give a guarded prognosis rather than an hopeless one until this time. During the period of trial and conservatism, most patients with myelomeningocele die of intercurrent respiratory tract infection, meningitis, ascending urinary sepsis or other maladies that result from concomitant malformation of other systems. The survivors of this group then present serious orthopedic problems. Plastic repair of the neural axial defect becomes necessary in order that appliances for the paraplegia may be worn with safety and comfort. Much of the orthopedic burden can be lightened by calling in the orthopedic consultant early in order that deformity by contracture may be avoided.

Preparation of an unusually wide field in operations for meningocele is necessary in many instances because of the frequent use of relaxing incisions to achieve closure without strain. The skin and protuberance may be prepared with any suitable chemical solution, preferably colored, in order to be certain that no area remains untouched. This is particularly important in view of the crevassing of these masses. It is well to have the patient's head slightly dependent throughout the procedure so that undue loss of spinal fluid by dependent drainage may be avoided. An elliptical incision running transversely to the neural axis and carried down to the deep fascia is usually more practicable than the vertical type. The skin and superficial fascia are mobilized by dissection up to the point of emergence of the neural sac from the bifid defect. Opening of the dura at a point obviously free from neural structure permits inspection of the contents with less chance of damage. At this point, a true distinction between meningocele and myelomeningocele is possible for the first time. Neural elements ending blindly in the wall of the sac are sacrificed, and presumably intact structures are returned to the neural canal. In resecting the sac, due care must be given to the preservation of sufficient dura for water-tight closure. Ingraham and Ham-
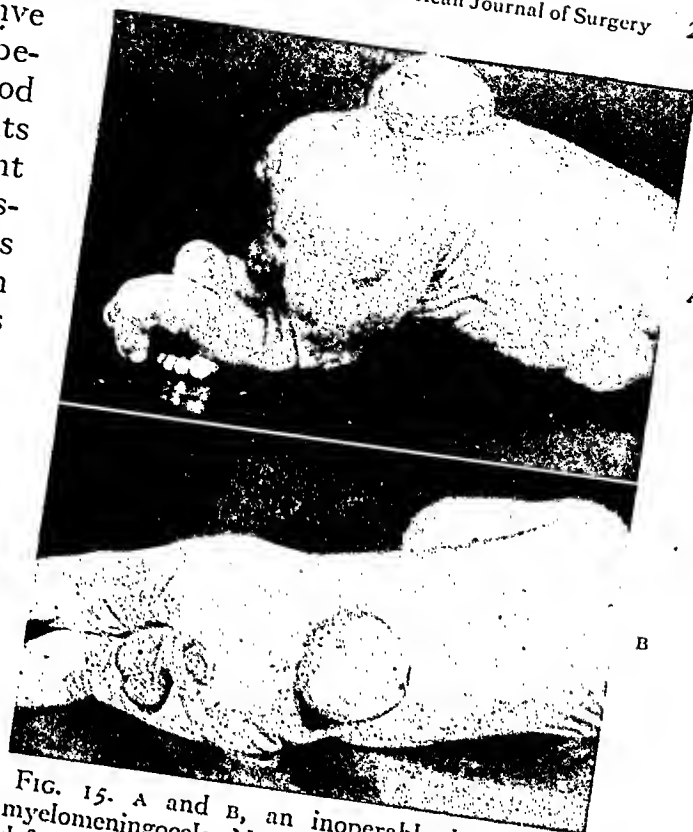
FIG. 15. A and B, an inoperable lumbosacral myelomeningocele. Note relaxed rectal sphincter, deformity of the legs and neural elements presenting on the surface of the lesion.

lin[25] advise that no attempt be made at bony reconstruction at this time. It may be feasible to reinforce the dural closure with flaps of fascia elevated from the sacrospinalis musculature, if in so doing, the procedure is not unduly prolonged and compression brought to bear on the contents of the reconstructed thecal sac. In large protuberances, in which the spinal canal is too small for the replaced neural elements, a reconstructed meningocele is created and relative safety achieved by closure of sound superficial fascia and skin above it. Not infrequently, a relaxing incision, such as the one Davis[26] attributed to Dieffenbach is useful in effecting closure without tension. (Fig. 16.)

Recently it has been the practice of this clinic to use a sulfonamide emulsion dressing surmounted by dry gauze and maintained by elastoplast adhesive. Lightweight rubber sheeting anchored by adhesive at the level of the apex of the intergluteal fold and carried forward over the entire dressing forms a further barrier against excretory contamination. The patient is

FIG. 16. A, photograph of a patient, eight months of age, with a large myelomeningocele.

moved from the operating table to a Bradford frame and secured in the prone position with padded restraints. A pliofilm covering of the frame prevents skin irritation. If there is any suspicion of incompetence of closure,

FIG. 16. B and C, diagrammatic drawings of the V-Y relaxing incision used to effect closure in this type of case.

the frame should be tipped slightly downward in order to reduce any hydrodynamic strain on the suture line. Vigilant nursing is required to observe cerebrospinal fluid leakage, decubitus formation and excretory contamination until the wound is thoroughly healed. Leakage occasionally occurs in spite of meticulous closure, possibly as the result of a change in cerebrospinal fluid dynamics created by operation. In this event, immediate corrective measures must be undertaken, such as resuturing of the wound at the site of fistula and relief of intracranial pressure by repeated ventricular taps. Should this not prove sufficient, a system of closed drainage of cerebral spinal fluid by either the lumbar or ventricular route is indicated. The pressure gradient can be controlled by a method described by Ingraham and Campbell.[27] Unless there is leakage or sepsis, the wound is best not examined until the eighth day. Sutures are usually removed between the eighth and tenth days. Urinary retention as a complication is handled by tidal drainage as advocated by Munro[28] with modifications made in the apparatus to adapt it to the age group in question.

CRANIUM BIFIDUM

Cranium bifidum corresponds with spina bifida and arises by the same process. Meningoceles occur in conjunction with cranium bifidum. Encephaloceles correspond with myelomeningoceles and arise as the result of faulty induction of ectoderm and mesenchyme.

INDICATIONS FOR SURGICAL INTERVENTION

Selection of patients for surgery in cranium bifidum and associated deformities is

FIG. 17. These photographs are of the same patient: A, four and one-half weeks old; B, postoperative photograph at six and one-half weeks.

based upon the same considerations that were outlined for the spina bifida group. Abnormalities of neurulation form the embryologic basis for judging what patients are suitable for surgical readjustment of congenital malformation. In this way, operation will not be needlessly offered to those with uncontrollable hydrocephalus, feeblemindedness and blindness due to basic malformations of cerebral structures. The age of choice for operation is twelve to eighteen months, as is the case with the spinal group. However, earlier consideration must be given to individuals threatened by meningitis from rupture or incipient ulceration of the covering of the protrusion. If the infant appears to be normal in all other respects, it is worth chancing future development by operating as early as the first day of life. In general, any severe mental retardation in the first twelve to eighteen months of life contraindicates surgery, as does progressive hydrocephalus, blindness or signs of severe deformity in other systems. When the decision cannot be made by history, physical and neurologic examination, pneumoencephalography by the lumbar or ventricular route is indicated. Gross bizarre malformation of the intracranial struc-

tures, outlined by this means, will make the final contraindication.

Simple cranium bifidum is rare in this clinic. Ingraham and Swan,[21] in 1943, in reporting the site of eighty-four cases of encephalocele and cephalic meningocele showed that sixty-three of them were occipital (Fig. 17), as opposed to five nasal, six frontal, nine parietal and one nasopharyngeal. The higher rate of true encephalocele formation, as opposed to meningocele in the occipital region is interesting because this is the first part of the neural axis to close. It was also their experience that the prognosis was poorer when an encephalocele was encountered.

Surgery of simple cranium bifidum consists of plating the bony defect with tantalum in order to protect the underlying brain from direct trauma to which all children's skulls are subjected when they are learning to walk and play. (Fig. 18.) Fixation with tantalum wire rather than with pegs is desirable since the calvarium is so thin. For this same reason, an acrylic inlay is not advised.

Excision and repair of meningoceles or encephaloceles along the vault of the calvarium is not a difficult procedure if the position of the venous sinuses of the brain is constantly kept in mind. The incision

FIG. 17. C and D, at eight and one-half years of age. The lesion removed at operation was a definite encephalocele which contained neural elements. This patient does reasonably well in school.

must be adapted to the position, size and shape of the defect in order that a water-tight dural closure may be effected and reinforced by firm galeal and skin closure. On the other hand, repair of some frontal (Fig. 19) and nasopharyngeal encephaloceles requires formidable intracranial surgery in order to effect an enduring water-tight closure in the region of the cribiform plate. Ingraham and Matson,[29] in 1943, described the successful handling of a case in detail. Dandy's description of the operative treatment of orbital meningocele published in 1929[30] is classic.

ARNOLD-CHIARI MALFORMATION

During the last twelve years, many excellent reports have appeared concerning the Arnold-Chiari malformation and its rôle in the production of hydrocephalus as a complication of meningocele and myelomeningocele formation. This discussion will not touch upon the origin of malformations such as Bucy and Lichtenstein[31] have re-

cently reported. Their patient showed no evidence of mesenchymal disturbance, such as platybasia or spina bifida.

In brief, the Arnold-Chiari malformation consists of downward displacement of the cerebellum and brain stem through the foramen magnum. Ascent of the cord occurs as the result of traction exerted by the relative fixation of the cerebellum and other components of the hindbrain within the calvarium, when the growth of the cord lags behind the more rapid growth of its supporting structure, the vertebral column. It is reasonable to suppose, therefore, that firmer fixation elsewhere along the neural axis, such as exists with meningocele or myelomeningocele might so reverse the direction of force exerted by the discrepancy of normal growth of the neural and spinal elements, that herniation of the posterior inferior portions of the cerebellum, hindbrain and adnexae results (Fig. 20), as well as reversal of the angle of the spinal nerve roots. (Fig. 21.) As this herniation is enhanced by growth, the constriction at the foramen magnum increases, with consequent obliteration of the subarachnoid space. Thus, passage of cerebrospinal fluid upward to the field of absorption in the neighborhood of the arachnoid villi
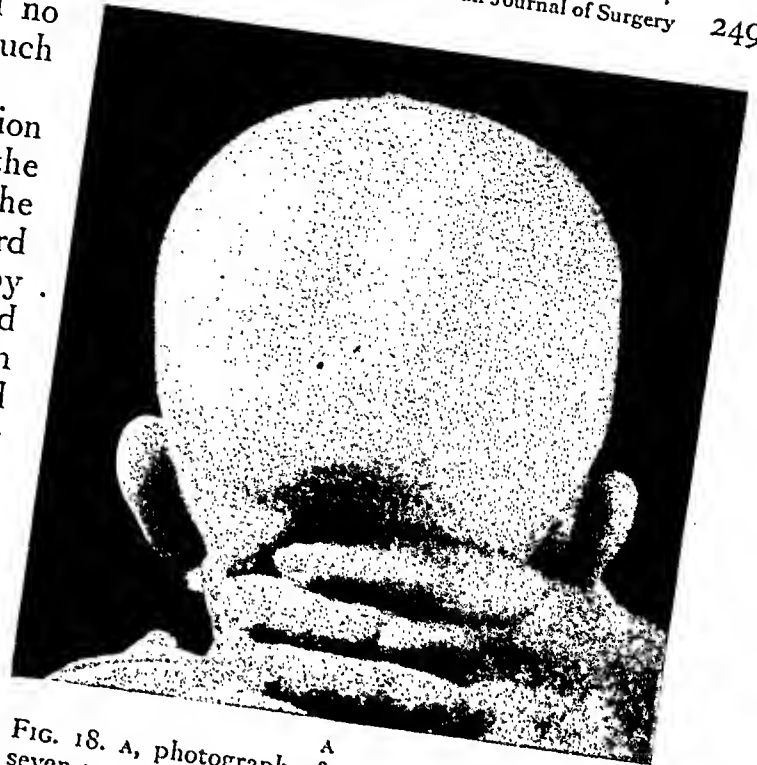
FIG. 18. A, photograph of a patient, aged one year, seven months, born with a simple cranium bifidum.

is prevented. Russell and Donald[32] drew attention to the hindbrain malformation as a possible cause of internal hydrocephalus. These authors suggested that decompression of the spinal cord at the foramen magnum might lead to cure. Penfield and Coburn[33] three years later advanced the theory that fixation of the cord at the site of the bifid spinal column might produce
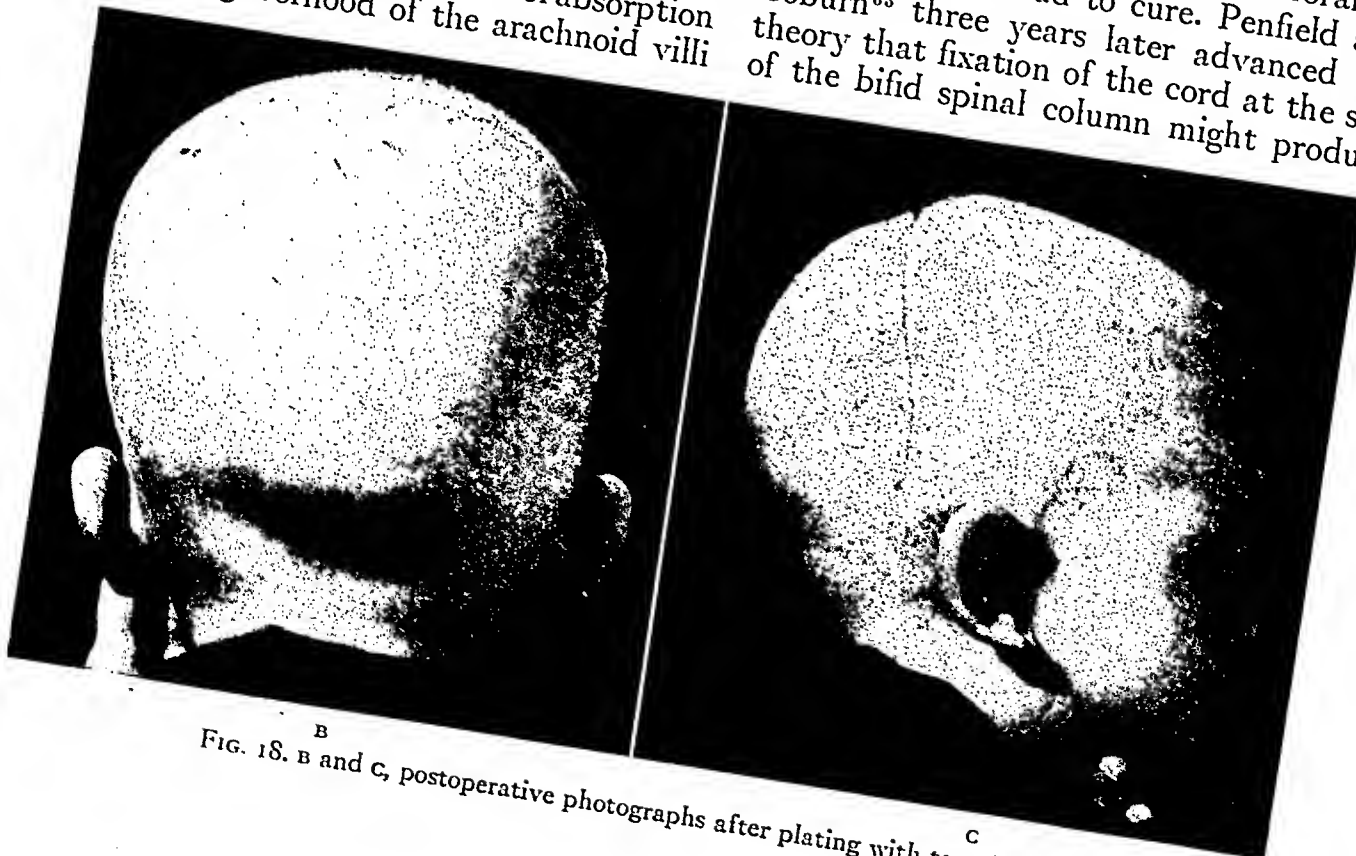
FIG. 18. B and C, postoperative photographs after plating with tantalum.

A

B

FIG. 19. A and B, these photographs show a patient with a frontal enecphalocele.

FIG. 20. Saggital section through the brain of a patient born with a myelomeningocele. Note the hydrocephalus, microgyria, and downward displacement of the inferior portion of the cerebellum and other hindbrain structures (Arnold-Chiari malformation).

Coburn, was applicable in all instances. Ingraham and Scott[35] reported the finding of microgyria in all of twenty cases studied in this clinic with Arnold-Chiari malformation associated with myelomeningocele. This finding is an important contribution to the list of other associated congenital malformations of ectodermal origin, such as stenosis of the Aqueduct of Sylvius, hypoplasia of the cerebellum, hydromyelia and diplomyelia. Associated defects of mesenchymal origin are frequently present in the upper cervical and basio-occipital region, such as fusion of cervical vertebrae (Klippel-Feil syndrome), spina bifida occulta, or platybasia of varying degree. Ingraham and Scott[35] suggested that microgyria in conjunction with myelomeningocele and the Arnold-Chiari malformation, constituted a clinical entity representative of a widespread defect. This is a tenable concept when consideration is given to the slight degree of differentiation of structures from which the cerebral hemispheres, cerebellum and fourth ventricle, are elaborated at the time that abnormal neurulation makes possible the formation of a low spinal myelomeningocele. In the late somite embryo, the anterior neuropore is closed on the twenty-fifth day, at which time the cerebral vesicles, primordia of the cerebellar hemispheres are but slight lateral dilatations of the prosencephalon. The fourth ventricle and primordium of the

the malformation by traction on the hindbrain. List[34] raised the question whether a primary developmental deformity of the hindbrain would not better explain the structural aberration noted in many cases of the Arnold-Chiari malformation. List did not believe that the purely mechanical theory of origin, offered by Penfield and

cerebellum have not been formed at this time in the rhombencephalic region. Therefore, any change of sufficient magnitude to derange morphogenetic movement in the region of the blastopore with resulting abnormal neurulation and ultimate myelomeningocele formation at one point, may possibly interfere with induction elsewhere. Hence, microgyria of the cerebrum, hypoplasia of the cerebellum and other associated anomalies may be interpreted as manifestations of malinduction possibly resulting from interference with organizer substances of a higher degree, or the competence of tissue stimulated by them.

Lichtenstein[36] has shown that the Arnold-Chiari malformation may be a reasonable mechanical cause of hydrocephalus noted at birth in patients with myelomeningocele formation. If this occult deformity is present in the posterior fossa of a patient born with an apparently normal cerebral spinal fluid circulation, who, coincidentally, has a meningocele or myelomeningocele, it is likewise justifiable to suppose that it may be the cause of hydrocephalus occurring at any time after birth. The sudden onset of hydrocephalus following repair of a meningocele or myelomeningocele was attributed by Penfield and Cone[37] to loss of an absorptive mechanism contained within the sac. It, however, has been the experience of this clinic that preservation of the sac has not prevented hydrocephalus. The presence of the Arnold-Chiari malformation has been demonstrated repeatedly by this neurosurgical service in patients operated upon for meningocele or myelomeningocele who have developed acute hydrocephalus during their immediate post-operative course. Therefore, it seems reasonable to conclude that patients of this type possess a narrow margin of safety because of an extensive degree of posterior fossa deformity. The shift in cerebrospinal fluid dynamics caused at the time of opening the meningeal sac during operation may cause further downward displacement of the already herniated portions of the hindbrain. Under these circumstances, con-

Fig. 21. Ventral view of the spinal cord at autopsy performed on a patient with an Arnold-Chiari malformation associated with a thoracic myelomeningocele. Note the upward course of the cervical nerve roots, diplomyelia and associated midline bony anomaly.

striction of the subarachnoid space above the foramen magnum may be so firm as to block the ascent of fluid and cause hydrocephalus. Operation with the head slightly dependent is advisable on the grounds that it may prevent this chain of events. It follows then that posterior fossa exploration is indicated in patients who develop hydrocephalus at any time following repair of a meningocele or myelomeningocele, if the condition of the patient's extremities, sphincters and degree of hydrocephalus is

compatible with a relatively normal existence. Occasionally, a patient will be brought to the surgeon for the first time with a meningocele or myelomeningocele and hydrocephalus of such degree that, if abated, normal mental development might ensue. If the extremities and sphincters in such a patient are intact and the spinal lesion favorable for repair at a later date, posterior fossa exploration may be done prior to spinal surgery.

Exploration of the posterior fossa can be carried out through a midline incision. Wide excision of the occipital bone and the foramen magnum is important. The extent of cervical laminectomy is indicated by the lowest limit of herniation of the cerebellar tips. It is the experience of this clinic that anomalous venous channel formation in the suboccipital dura is as constant a finding in the operating room as microgyria in the postmortem room in patients with the Arnold-Chiari malformation. Therefore, it is advisable to exercise much care in the opening of the dura. Once the extent and nature of the anomaly has been estimated by exposure, adhesions are freed insofar as possible. If the herniated cerebellar tonsils are firmly fixed to the medulla and upper cervical cord, it is inadvisable to dissect them free, for fear of compromising the intrinsic circulation of these regions. Usually, lysis of adhesions on either side of the brain stem permits a free flow of spinal fluid from the region of the distorted fourth ventricle into the lateral recesses. However, if circulation cannot be re-established along the normal route, the by-passing procedure, originally described by Torkildsen,[38] for obstruction of the Aqueduct of Sylvius, may be employed. This consists of connecting the subarachnoid space of either lateral recess with the corresponding lateral ventricular system by means of a catheter. The catheter is first introduced into the lateral ventricle through a burr hole and then led subcutaneously to the lateral recess via the suboccipital decompression. Pains must be taken to see that the catheter is syphoning before establishing it in the

subarachnoid space, lateral to the cerebellar hemisphere. When these procedures have been of no avail, partial extirpation and coagulation of the choroid plexus of each ventricle may be undertaken. Occasionally, this procedure, designed to reduce the production of cerebrospinal fluid, is sufficiently effective to bring about a balance between its absorption and production.

TERATOMA AND TERATOID FORMATIONS ALONG THE NEURAL AXIS

A sacrococcygeal teratoma is usually a large protuberant mass which greatly deforms the buttocks. Displacement of the orifices of the perineum may signal retrosacral extension of such degree that the bladder and rectosigmoid portion of the colon may be involved to the point of mechanical danger. Aside from the mechanical indications for operation, there is the constant threat of potential malignancy arising from epithelial elements developed within such structures. Lisco[39] in a review of the literature and report of two cases, points out that malignant degeneration in teratomas arising at this site, is as likely as elsewhere. MacCallum[40] believes that adenocarcinoma is the most common type of malignancy occurring in such lesions. This alone is an indication for early and complete resection, as has been the policy of this clinic for over thirty-one years.[41] Complete resection may usually be effected in one stage. When the lesion is very large in proportion to the total size of the patient and contains an extensive retrosacral component, it has been found practicable to carry out removal in two stages, the retrosacral dissection being undertaken as the second procedure. This permits use of the lithotomy position and greater ease in dissection in the retroperitoneal space. The actual surgical procedure is formidable only in that the mass is usually large in proportion to the patient, and liberally supplied by blood vessels. Varying degrees of spina bifida and meningocele formation accompany sacrococcygeal teratomas. Therefore, it is important to achieve a water-tight

dural closure and preservation of any herniated elements of the cauda equina. The decision for or against radiation should rest upon the result of a careful and searching pathologic study correlated with the completeness of the removal.

INTRACRANIAL AND INTRASPINAL TERATOMAS AND TERATOID TUMORS

Intracranial and intraspinal teratomas and teratoid tumors differ from the sacrococcygeal group in that they do not have constancy of position or develop, as a general rule, such a high degree of cellular differentiation. Craniopharyngiomas, pinealomas and chordomas will be omitted from this discussion because of the individual problems they present. The signs and symptoms produced by intracranial and intraspinal teratomas and teratoid tumors indicate the structures and levels involved. Ingraham and Bailey[16] conclude from a detailed study of a series of such tumors that congenital defects associated with signs of an expanding lesion suggest but do not firmly establish, the correct diagnosis preoperatively. Their study reveals that midline ectodermal defects occasionally overlie posterior fossa teratoid tumors, but more commonly exist in association with intrapsinal tumors. In some of their patients with this latter type of lesion, they were able to demonstrate roentgenographically a bifid defect. This was not their experience with the intracranial group.

SURGICAL MANAGEMENT

Exploration is indicated by signs of a space-occupying lesion. The decision as to the correct approach in the intracranial group rests with the correlation of data obtained by neurologic and roentgenographic examinations. Ventriculography is used, unless a dermal sinus points to the lesion, as has been the case with several patients operated upon in this clinic, who had teratoid lesions in the posterior fossa. The presence of spina bifida, with or without an associated ectodermal defect, correlated with neurologic and spinal fluid
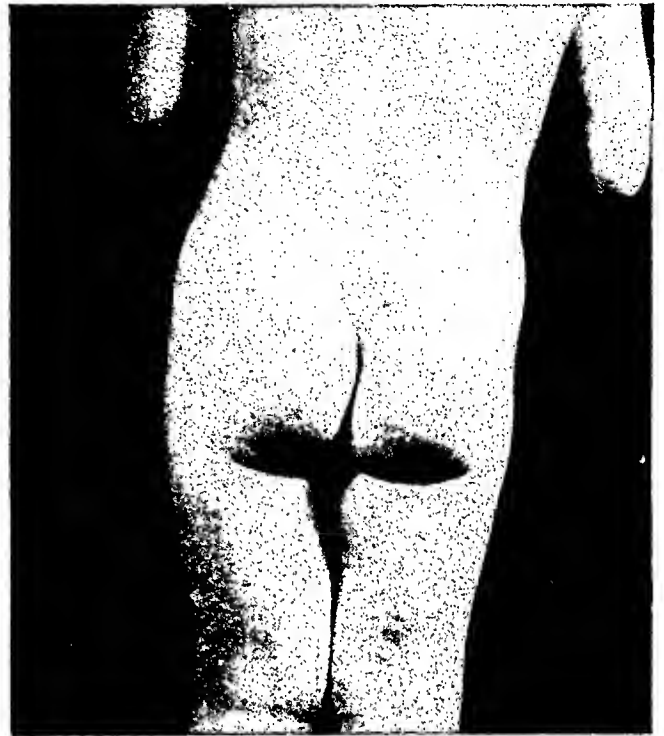
FIG. 22. This photograph shows the surface opening of a persistent accessary neurenteric canal surrounded by a collar of abnormal fatty tissue in a child aged two years, eleven months.

studies, usually gives sufficient localization to permit exploration without myelography. The operative result and ultimate prognosis will be determined entirely by whether the lesion is discrete or infiltrative, composed of benign or malignant elements, and if it is so situated that removal can be effected without damage to surrounding neural structures. Malignant lesions can be dealt with only by decompression and radiation after biopsy. Certain benign intramedullary spinal lesions which would not permit complete resection because of their intimate application to the cord have, in Ingraham and Bailey's[16] experience, been benefited for more than five years by a partial resection and aspiration. Preoperative infection of teratoid lesions by bacterial invasion along an associated dermal sinus, greatly reduces the chances of a favorable outcome with a benign lesion. However, with the aid of such chemotherapeutic agents as sulfadiazine, streptomycin and penicillin, removal of an infected intraspinal dermoid has recently been accomplished in this clinic. Grievous past experience with atypical meningitis, de-

FIG. 23. A, this photomicrograph reveals the tract of the persistent accessory neurenteric canal surrounding skin and superficial fascia. Hematoxylin and eosin stain: magnification 15 diameters.
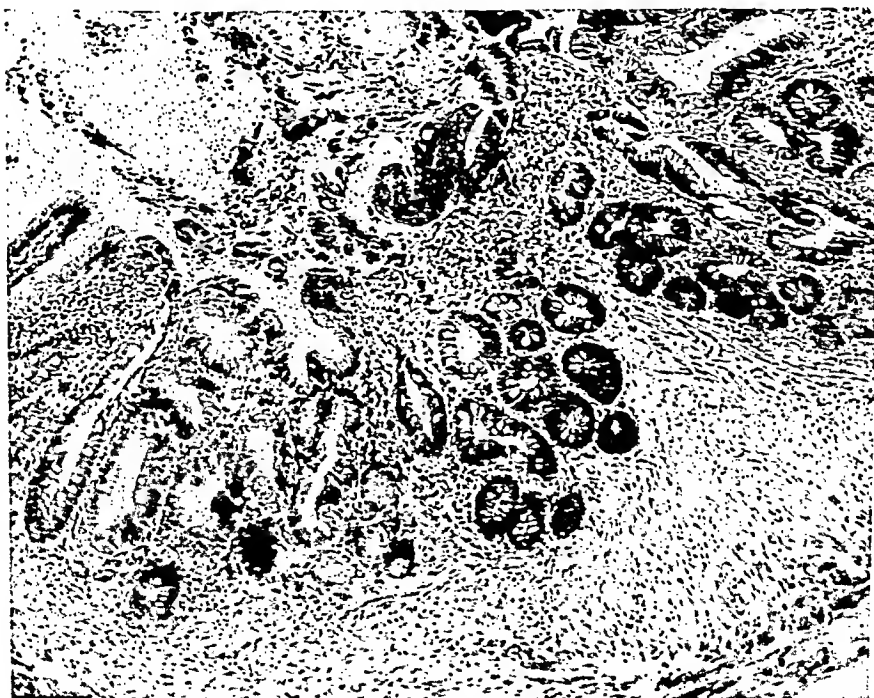
FIG. 23. B, further magnification, 130 diameters, reveals intestinal mucosa and muscular coat of the rudimentary intestinal tract.

veloping as the result of the extension of infection to the subarachnoid system from a septic teratoid lesion, has led to the policy that the presence of a midline dermal sinus, other than a pilonidal sinus, is sufficient indication for operating without waiting for neurologic signs to develop.

PERSISTENT REMNANTS OF THE NEURENTERIC CANAL

A theoretic basis for persistent neurenteric canal remnants and their association with spina bifida was proposed before in the basic embryologic discussion. This is an extremely rare condition in The Children's Hospital series (two cases). Spina bifida without neurologic deficit was present in both cases. The presence of an eccentrically placed fistulous tract (Fig. 22) in the lumbosacral region which might serve as a potential route for the spread of sepsis to subarachnoid system, was the indication for operation upon each patient. Operation consisted of block excision of the superficial ectodermal defect and surrounding skin, followed by mobilization of the tract down through the spina bifida to its point of emergence from the dura. In both patients a watery discharge from the fistulous opening onto the skin had been noted. In spite of this fact, no spinal fluid appeared on the transsected surface of the proximal stump of the tract. Nevertheless, a transfixing suture was placed about it to assure watertight closure of a potential connection to the subarachnoid space. It was then presumed that the watery discharge was a glandular secretion rather than cerebrospinal fluid. Both patients made an uneventful recovery. Microscopic examination of each specimen revealed normal skin and adnexae surrounding a tract lined by enteric mucosa, supported by muscularis and connective tissue elements, such as exist along the gastrointestinal tract. (Fig. 23.) These lesions may be teratomas, but it seems preferable to follow the hypothesis of Bremer[5] and to consider them as evidence of persistent neurenteric canal remnants displaced by an early aberrant morphogenetic movement.

SUMMARY

A review of relevent experimental embryology indicates that congenital anomalies of the neural axis are the result of disorders of neurulation. The surgical principles involved in the treatment of spina bifida, cranium bifidum and associated anomalies such as meningoceles, myelomeningoceles, encephalocles and the Arnold-Chiari malformation are discussed as well as teratomas and teratoid tumors arising along the neural axis. A correlation of embryologic and neurologic data is used in the selection of patients for surgery and the time for and type of operation to be performed on each individual case. The genetic basis of origin of congenital anomalies of the neural axis is considered as well as environmental factors that may cause disorders of neurulation. Hyperthermia in the mother occurring between the third and fifth week of gestation is suggested as a possible cause of such disorders.

The surgery of congenital anomalies can be more intelligently carried out when consideration is given to the embryology of the system concerned.

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CORTICAL EXTIRPATION IN THE TREATMENT OF INVOL-UNTARY MOVEMENTS

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DURING the past fifteen years there has been an increasing interest and understanding of the problem of the surgical treatment of such abnormal involuntary movements as choreo-athetosis and the various tremors. Although Horsley[6] and others had operated upon a number of such patients many years before, interest in the problem had largely died down until the publication by Bucy and Buchanan[4] in 1932. In spite of numerous publications since then concerned with actual surgical procedures in human cases, animal experimentation and with theoretical considerations, the field is still one of active investigation, discussion and indecision. It is not surprising that this is so. Except for a very few instances involuntary movements comparable to those observed so commonly in human disease have not been produced in the experimental animal. Recent experiments by Ward, McCulloch and Magoun[8] have given cause to hope that this deficiency may soon be repaired at least in part. Nevertheless, this is still little more than a hope and thus far we have been dependent for our knowledge of the pathologic anatomy, pathologic physiology and the best means of therapy upon the study of those human experiments which nature provides for us. Suitable cases of this type are not too numerous and progress has been correspondingly slow. Thus far there is no surgical procedure which can be regarded as a satisfactory solution to the problems of these unfortunate people. *All operations thus far devised for the relief of abnormal involuntary movements must be regarded as distinctly experimental; all of them are unsatisfactory in major ways; no one of them has yet reached the stage where it can generally be recom-mended as a form of treatment routinely in any one of these conditions.* All operations for the relief of these conditions should be the occasion for intensive study. Much more knowledge is needed. Only by very critical, careful, objective observation and evaluation can we arrive at the true facts, the only sound basis for understanding the disease and planning intelligent therapy.

DEFINITIONS

The abnormal involuntary movements with which we are primarily concerned fall into two large groups: One of these is characterized by bizarre, irregular, unpredictable, non-repetitive types of movement which are commonly denominated by the term *choreo-athetosis*. These movements may be quick, slow or intermediate. Although they generally involve the peripheral part of the extremity more than the proximal, this is not invariably true; and in hemiballismus, which otherwise falls in this category, the movements are generally greater at the proximal joints than at the distal joints. The upper extremity is generally more severely involved than the lower but the degree of involvement is variable. In many instances the extremities are predominantly involved but in dystonia musculorum deformans or torsion dystonia, which is certainly a form of choreo-athetosis, there is generally marked involvement of the trunk as well. In general the movements are not only purposeless in fact but do not even have the appearance of purposeful movements. This is in contrast to the quick, jerky movements of Sydenham's chorea, which although involuntary, abnormal and purposeless so far as the individual is concerned, have, as Wilson repeatedly pointed out, the

appearance of purposeful movements. They are well coordinated muscular movements of the type which the patient might make voluntarily. This in general is not true of the movements of choreo-athetosis.

The second type of involuntary movement under consideration is the *tremors*. Although there are three different types of tremor which can be defined, they are not necessarily distinct entities. Many patients will present two or even all three types of tremor. Tremor may be divided into tremor at rest, static tremor and action tremor. Tremor is the involuntary occurrence of regular rhythmic movements in which the same movement is performed over and over again usually at a rate of from four to eight per second. *Tremor at rest* occurs in the supported extremity which is not involved by voluntary muscular contraction. Such tremors are characteristic of paralysis agitans or parkinsonism. It has been purposelessly argued that such tremors are not really tremor at rest as they are not present when the muscle is most completely at rest, i.e., during sleep. However, like all descriptive terms, this one has meaning which is capable of definition, of being readily understood by all who are familiar with the phenomenon and of being useful. The mere fact that the term cannot be stretched to cover some situation for which it was never intended is not an argument for introducing a new and unfamiliar term for one which is well established, or for denying the existence of the well recognized facts. The other two types of tremor might well be grouped together as intention tremors since both occur in muscle which is voluntarily innervated. The *static tremor* occurs in muscles which are engaged in supporting a part of the body stationary. *Action tremor* occurs in muscles which are engaged in moving a part of the body. Such tremors are very familiar by personal experience to most of us as they need not be dependent for their appearance upon the presence of organic disease. Both frequently occur as the result of a common functional disturbance, muscular fatigue. However, in the present discussion we are concerned with the more serious and persistent form of intention tremor which is characterized by actual alterations within the structure of the nervous system.

PATHOLOGY AND ETIOLOGY

These abnormalities of movement may arise as the result of various types of disease processes. Thus *infections* such as the infantile encephalitis may give rise to choreo-athetoid movements; the virus encephalitides may give rise to a parkinsonian tremor or to choreo-athetosis; syphilis of the mid-brain may produce the picture of paralysis agitans. *Developmental deficiencies* of the basal ganglia may produce athetosis or torsion spasm. *Degenerative disease* by affecting the same areas may produce the tremor both at rest and in connection with movement which is characteristic of Wilson's hepatolenticular degeneration. Degenerative processes may also lead to Parkinson's disease and to the bizarre movements of Huntington's chorea. *Vascular disease* by destroying various parts of the basal ganglia may produce tremor both at rest and in association with voluntary movement, may produce choreo-athetoid movements or the violent uncontrollable movements of the entire extremity which is characteristic of hemiballismus. Severe cerebral *trauma* may occasionally produce changes within the basal ganglia of such character and severity as to lead to the appearance of involuntary movements such as these. However, trauma is much less often responsible than these other disease processes. It is of interest, however, that neoplastic diseases in this region rarely produce any of these abnormal involuntary movements.

It is obvious from the foregoing that the nature of the causative disease process bears little if any relationship to the type of involuntary movements which develop. Rather the nature of the movements is largely dependent upon what part of the nervous system is destroyed, rather than

upon the nature of the destroying agent. Our knowledge in this field is not yet complete but in general the following statements are in accord with the findings of most investigators. *Tremor at rest*, or parkinsonian tremor, is found in association with destruction of the globus pallidus, the substantia nigra or both. It is surprising how frequently lesions are found in these two anatomically connected but separate parts of the basal ganglia in cases of parkinsonism. In this same connection it is of interest that in the monkeys in which Richter produced a tremor, as well as the rigidity and slowness of movement characteristic of parkinsonism, by chronic carbon disulfide intoxication, the essential and constant pathologic changes were in the globus pallidus and the substantia nigra. *Intention tremor*, on the other hand, is seen most consistently in association with lesions of the nerve fibers arising from the dentate nuclei of the cerebellum and passing forward through the red nuclei to the thalamus, whence their impulses are relayed to the precentral motor cortex. *Choreo-athetosis* is usually seen in association with lesions of the putamen and caudate nuclei of the basal ganglia but lesions only in the globus pallidus or in the thalamus have also been described. *Hemiballismus* arises as the result of destruction of the subthalamic body of Luys.

PATHOPHYSIOLOGY

It must be obvious to anyone who will give the matter a moment's thought that, although these abnormal involuntary movements may arise as the result of the destruction of these subcortical nuclei, they cannot be directly produced by these structures which are gone. The movements can arise only as the result of the activity of some portion of the nervous system which is still present and active. It has been quite commonly accepted that these movements represent the abnormal hyperactivity of some portion of the nervous system which has been released from the normal controlling influences of those portions of the nervous system which have been destroyed. We are indebted to the late Kinnier Wilson for having drawn our attention most forcefully to the probability that the precentral portion of the cerebral cortex and the motor pathways arising from them constitute the nervous mechanism which is released and which is directly responsible for the production of these movements. In 1942, I developed at some length a hypothesis explaining how each of these structures in the subcortical nuclei might exert a controlling, restraining, inhibitory or suppressor influence on the precentral motor cortex. This was based upon the known facts of neuroanatomy and neurophysiology, many of which had only recently been elucidated by the late Dusser de Barenne[5] and his disciples. No new developments since that time have brought forth any evidence to controvert that hypothesis. On the other hand, that hypothetical explanation is by no means the only possible pathophysiologic explanation of the manner in which the nervous system operates to bring about these abnormal movements. It is, however, the most complete explanation that has been proposed. I shall not here again present the details of that discussion. Suffice it to say that there are anatomic pathways leading from the cerebellum, substantia nigra, globus pallidus, putamen and caudate nucleus by way of the anterior part of the lateral nuclear mass of the thalamus to the precentral motor cortex of the cerebral hemisphere. There is also physiologic evidence that in some cases at least the functional activity of these pathways is one of suppression or inhibition of the activity of the precentral motor cortex. It has been my belief that these various subcortical neural mechanisms exert a controlling influence on the precentral motor cortex and that when that influence is removed the precentral cortex responds by an abnormal nervous hyperactivity which produces these involuntary movements. The type of movements which develop will depend upon the nature of the

controlling influence which is removed and will, therefore, vary depending upon which subcortical mechanism is destroyed.

The precentral motor cortex is a complex cortical area which has been fully discussed in the author's monograph.[2] The two principal subdivisions which are concerned with the innervation of the skeletal musculature and the production of movement are the two areas, designated as area 4 and area 6. The types of movement produced by these two areas differ in several respects. The various subcortical mechanisms mentioned above differ in their connections with the precentral motor cortex. It seems likely that some of them exert greater influence on one portion while others are more concerned with other parts of this cortical area. The precentral motor cortex gives rise to two large fiber systems. They are the pyramidal tract which extends directly from the precentral region to the spinal cord and the extrapyramidal systems which are much more complex and extend from the precentral cortex to subcortical areas, whence their impulses are relayed to other portions of the nervous system, including the spinal cord. There is some imperfect yet highly suggestive evidence that that portion of the pyramidal tract which arises from area 4 of the precentral cortex (some 50 per cent arises elsewhere) is concerned with the production of tremor, both at rest and in association with voluntary movement; while other evidence indicates that choreoathetosis is produced by impulses which travel over the extrapyramidal fibers which arise from both areas 4 and 6. As yet we do not know which of these various extrapyramidal systems is so concerned nor do we know which of the subcortical nuclear centers is connected with these extrapyramidal fibers.

SURGICAL TREATMENT

We are concerned here only with excision of the cerebral cortex in the relief of abnormal involuntary movements. Other surgical procedures such as section of the anterior limb of the internal capsule or extirpation of the caudate nucleus have been discussed elsewhere in this issue (see Browder).

In my experience it requires a much more extensive extirpation of the precentral motor cortex to abolish the movements of choreo-athetosis than it does those of tremor. This is not surprising if our hypothesis is correct that the movements of choreo-athetosis are produced by the extrapyramidal fibers which arise from both areas 4 and 6 whereas those of tremor are produced by pyramidal fibers arising only from area 4. At operation it is my practice first to determine the representation of the upper and lower extremities in the precentral region by stimulating the exposed cortex with a sixty-cycle sine-wave electrical current. Only when this has been carefully done can the surgeon plan his extirpation intelligently. Various persons have reported failures from what they claimed to be duplication of the procedure which I have advocated. Study of their operative reports or of the brains of their patients who have subsequently died has often shown that the extirpation was far too limited or that it was not placed in the precentral motor cortex at all. Such errors could not be made if the operator had carefully outlined the representation in this region by electrical stimulation.

In endeavoring to relieve choreo-athetosis, it is my practice to remove the precentral gyrus and part of the frontal gyri lying anterior to it. Posteriorly the extirpation should extend down to the bottom of the central or Rolandic fissure, usually a matter of 1.5 to 2.0 cm. Anterior to the central fissure the extirpation need not be so deep as it is necessary only to remove all of the grey matter down to the subjacent white matter. Any remaining fragments of grey matter situated in deep-lying fissures can be readily removed with the suction apparatus. It has been my experience that the extirpation is most easily carried out by a subpial dissection as advocated by Horsley and Sachs, using a curved semi-

sharp instrument such as a small periosteal elevator. In this way there is minimal damage to the vascular system and to neighboring gyri. Large arteries and veins crossing the site of the extirpation are easily spared, thus reducing the possibility of infarction of tissues at a distance from the extirpation. In most cases of choreo-athetosis both the arm and leg are involved. It is best, therefore, to remove the representation of both the upper and lower extremities. The extirpation should extend not only up to the interhemispheric fissure but over the crest of the hemisphere and down the medial surface to the first sulcus. In those cases in which the hemisphere is very adherent to the superior longitudinal sinus and efforts to separate them results in troublesome bleeding, this part of the extirpation, at the crest of the hemisphere, is best carried out with suction even though this destroys the tissue from this small area and precludes subsequent microscopic examination. In those few cases in which the choreo-athetoid movements are restricted to the upper extremity the extirpation can be limited to the related area and the representation of the lower extremity spared. Such a procedure gives rise to considerably less neurologic deficit, and thus the patient is less handicapped.

Obviously such an extirpation results in a partial paralysis of the contralateral extremities. As has been pointed out elsewhere (Bucy)[2] the resulting paralysis is not complete as the extremities receive some innervation from other parts of the brain, notably the remaining portions of the precentral motor cortex on the side of the operation and that of the opposite cerebral hemisphere. When it is possible to spare the representation of the lower extremity, the degree of paralysis in the arm is far less than when both the "arm" and "leg" areas are removed. When the representation of both extremities is destroyed voluntary movements in the upper extremity are severely limited. Little if any useful movement is present in the hand and fingers while the amount of movement

increases proximally. In such cases the upper extremity is almost useless. For that reason no patient should be operated upon unless either the choreo-athetosis or the associated hemiparesis which so often accompanies it are so severe as to make the extremity practically useless prior to the operation. In every instance in which I have operated for the relief of choreo-athetosis the patient, both before and after the operation, has been happy to trade his uncontrollable involuntary movements for a greater degree of paralysis. No one of them has ever expressed any regret over his decision. It should ever be so.

Fortunately the degree of disability so far as the lower extremity is concerned is much less. This is true for two reasons. First the amount of bilateral representation in the cerebral hemispheres is greater for the lower than it is for the upper extremity. Therefore, the degree of paralysis which follows extirpation of the precentral motor cortex is less in the leg than it is in the arm. Second, the nature of the paralysis is such as to cause less disability in the leg than in the arm. As in the arm the paralysis is greater at the distal joints than it is at the proximal joints. Paralysis of the hand causes a much greater disability in a prehensile extremity like the upper one than does paralysis of the foot and ankle in a supporting member like the leg. Furthermore, as the paralysis is more or less spastic that, too, interferes with active function of the arm and hand whereas it is much less of a barrier to the proper function of the leg which is primarily concerned with serving as a rigid prop for the body. Although the upper extremity is rendered practically useless by such an operation I have never had a patient who was unable to walk following such an extirpation. Many of them are able to walk for miles and are engaged in occupations which require that they be on their feet a part of the time.

Because of the nature of the spastic paresis which of necessity follows this

operation, it of course is limited in its application to individuals who have choreoathetosis on only one side of their body or in whom it is so much more severe on one side than on the other as to make a unilateral operation and unilateral relief worth while. The operation is not therefore suitable for cases of double athetosis or in cases of dystonia muculorum deformans. Furthermore, the operation does not attack the basic etiology or pathology of the disease and does not therefore have any effect in preventing progression of the disease or in relieving other manifestations of the disease. It should not be undertaken in cases in which the condition is obviously progressive or in which other symptoms are so severe and prominent as to make abolition of the involuntary movements of little value.

I should like to make it clear here that I have always found it most difficult to assess "improvement" in so far as mere lessening of the involuntary movements is concerned. The results of the operation described above are not mere improvement or reduction in the severity, extent or frequency of involuntary movements but the actual *abolition* of such movements. One should be content with no less. My observations would lead me to believe that where anything less than abolition has been obtained the operation has been incompletely or incorrectly performed.

As stated above my experience indicates that an extirpation limited to the precentral gyrus (area 4) suffices to abolish tremor. Again the extirpation should be performed subpially. It should extend, posteriorly, to the depth of the central fissure. It should include all of the representation of the arm or of the arm and leg depending upon the extent of the disease. Nothing in my experience leads me to believe that it is possible to abolish tremor by any procedure which does not interrupt the pyramidal tract or destroy the origin of that portion of it which arises from the precentral gyrus. At one time it appeared that an extirpation limited to the posterior one-half of the precentral gyrus (Bucy)[5] would suffice to abolish tremor. Such an extirpation removes the Betz cells from which the largest fibers of the pyramidal tract arise. Certainly such an extirpation has a profound effect upon the tremor but it will not abolish it completely and permanently.

Obviously even this limited operation results in a serious contralateral hemiparesis even though it is not quite as severe as that which follows the more extensive operation necessary to relieve the movements of choreo-athetosis. Therefore, it should not be undertaken unless the tremor is so severe that a hemiparesis will be welcome in its place. It must be borne in mind that here we are dealing with a profound difference between the two conditions. Choreo-athetosis is commonly associated with some degree of hemiparesis. Therefore, some increase in that condition, especially when it is attended with abolition of the very disturbing involuntary movements, hardly adds to the disability of the patient. With tremor the situation is different. It is rarely associated with any degree of paralysis of the affected extremity. The extremity is often a most useful member. One is, therefore, most hesitant to produce a paralysis where none exists. Only in the most aggravated cases should such a course be considered. Again, as with choreo-athetosis, the operation will not affect the progress of the disease. Most cases of parkinsonism are definitely of a progressive character and are, therefore, not suitable for such surgical treatment. What is gained by stopping the tremor in a man's left arm and rendering it useless, if next year or the year after the right arm will be similarly involved by tremor? It must also be remembered that the other symptoms of parkinsonism are not remedied by such an operation. In many cases the abnormalities of posture and of gait, the slowness and the poverty of movement, the muscular rigidity and the generalized aching are more annoying than the tremor itself. These symptoms will not

be relieved. In fact the rigidity will have added to it some measure of spasticity so that the stiffness of the extremities will most certainly be more marked after the operation than before. Furthermore, in my opinion the operation should be restricted to relatively young persons who are able to withstand the operation and, more important, are able to make the postoperative adjustments and to re-educate and develop the paretic extremities to the fullest possible degree.

I have tried to make it clear that in my opinion cortical extirpations, although of great interest in understanding the mechanism of tremor and of paralysis agitans, are of very limited usefulness in the treatment of parkinsonism. Only occasionally does one see a unilateral, non-progressive parkinsonism in a relatively young patient to whom the tremor is the greatest disability and for whom the tremor is so severe that both he and his physician consider it wise for him to exchange it for a partial paralysis of the affected extremities. Certainly not more than one in five

hundred of the people afflicted with this disease could so qualify. Most of those for whom the operation is suitable are atypical cases, such as those post-traumatic and luetic cases that I have operated upon and reported.

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SECTION OF THE FIBERS OF THE ANTERIOR LIMB OF THE INTERNAL CAPSULE IN PARKINSONISM*

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IT seems reasonably well established that the tremor, and in some instances, the rigidity of Parkinson's disease may be favorably modified by (1) excision of a part of the precentral motor cortex, (2) division of a part of the anterior limb of the internal capsule, (3) section of the pallidofugal fibers or (4) section of the corticospinal fibers in the upper cervical cord. Removal of the head of the caudate nucleus on one side has no effect whatsoever on the tremor or rigidity of parkinsonism.

The present communication is primarily concerned with the technical features of the capsular operation and the results to be expected following section of an appropriate amount of the fibers coursing through the anterior limb of the internal capsule. This surgical procedure is still considered to be in the experimental stage and should be carried out only on a carefully selected group of patients with parkinsonism. If properly executed, the tremor can be abolished without paralysis. Moreover, the results following this operation are superior to those that I have had the opportunity to observe in patients submitted to any of the other three above enumerated procedures.

CAPSULAR OPERATION FOR PARKINSON'S DISEASE

The operation is conducted under local anesthesia without pre-operative medication. The subject is placed supine on the table and the drapes so arranged to give a clear view of the extremities contralateral to the side of the brain to be surgically exposed. These extremities should not be restrained or otherwise encumbered, especially the upper one. A small bone flap is outlined and turned down in the mid-frontal region—fashioned to expose the falx and the superior part of the frontal lobe. After reflecting the dura and gaining proper orientation, a 2.5 to 3 cm. incision is made in the cortex just anterior to the premotor area and 3 cm. from the falx which it parallels. (Until well acquainted with the approach to the lateral ventricle it is well to locate the anterior aspect of the ventricle with a cannula which is left in place as a guide.) The incision is carried into the most anterior aspect of the lateral ventricle, thus exposing the head of the caudate nucleus. (Fig. 1.) The upper half of this structure is removed either by suction or scoop, thereby exposing the inner fibers of the anterior limb of the internal capsule. (Fig. 2.) The most anterior of these fibers in the corona radiata curve in rainbow fashion from forward aft to enter the capsule. Using a blunt right angle hook, the arm of the hook being 0.8 cm. in length, section is begun at the rostral end of the capsule and at a level just short of the termination of the corona radiata. (Fig. 3.) (A medium-sized blunt-end probe may be appropriately bent and used for sectioning.) Until familiarity with the operation is attained, it is best to section about 1 mm. of the capsular fibers at a time, then wait two or three minutes. During this rest period the surgeon himself makes observations regarding the amplitude of the tremor of the contralateral hand and the grip power is recorded. Slowly, bit by bit, the fibers are sectioned toward the knee of the capsule and observations as suggested are made and

recorded. As a point approximately 1.5 cm. anterior to the knee of the capsule is approached the patient not infrequently becomes difficult to arouse and only after rough prodding is he capable of cooperating. After five to ten minutes the drowsy

becomes active. Further sectioning of the capsular fibers up to a point approximately 1 cm. from the knee completely abolishes the tremor. This is not the end point of the operation. If further sectioning is not carried out, the tremor usually returns within
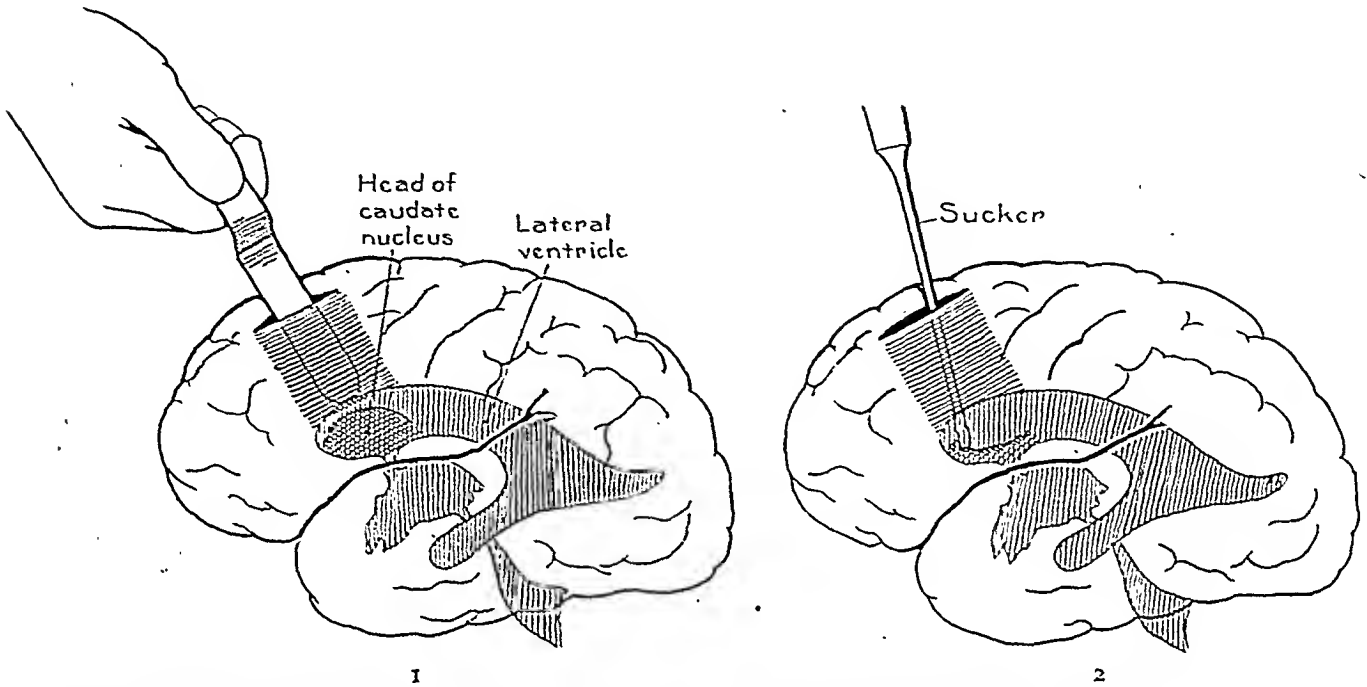
FIG. 1. Lateral view indicating approximate site of cortical incision and approach to the ventricle.
FIG. 2. Removal of part of the head of the caudate nucleus to expose the fibers of the anterior limb of the internal capsule.

state passes and the tremor which usually abates during the drowsy period again

a few weeks after operation. It is therefore important to carry the sectioning further and the operation is completed only after the production of a marked paresis of the hand. (Fig. 4.) In other words, the patient is just able to elevate the upper extremity
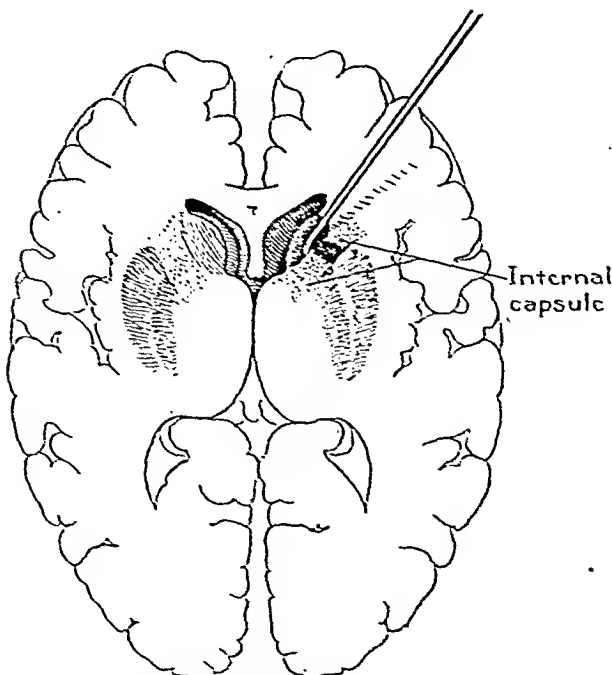
FIG. 3. Manner of sectioning the fibers of the internal capsule.
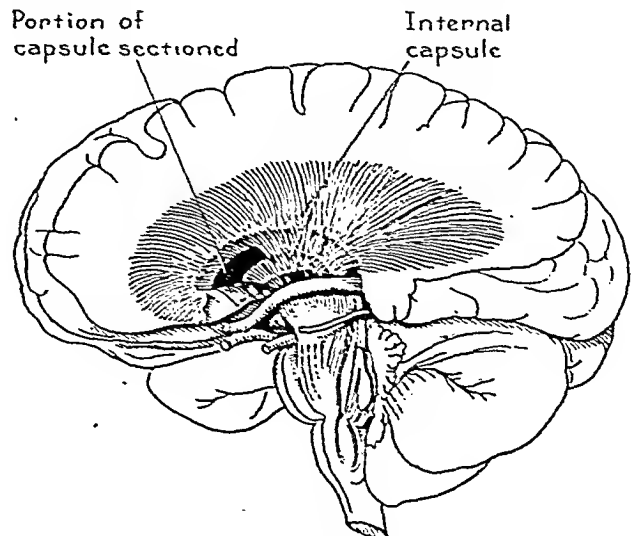
FIG. 4. Indicating approximate amount of fibers to be sectioned.

from his side and is only capable of slight flexion of the fingers without being able to grip the observer's hand. Examination of the lower extremity at this time will show that this part is equally paretic with the upper. Following complete hemostasis the wound is closed in the usual manner with layer silk. Immediately after operation the extremities affected by the procedure are relatively flaccid, paretic and Babinski's sign and the so-called confirmatories are present. The abdominal reflexes are abolished on the involved side. The patient is usually drowsy but easily aroused and cooperative. On the day following operation the hemiparesis is often more severe, gross movements of the involved parts, however, being preserved. There is a tendency for the patient to "sleep" when left undisturbed. Food and fluids are taken readily. Excessive sweating may be evident. A mild degree of fever prevails but hyperthermic states are uncommon. Gradually the motor power returns and by the tenth to twelfth day, when the patient is allowed out of bed, a mild to moderate hemiparesis is demonstrable. Slowly, ability to perform skilled acts with the paretic hand is regained. Forced grasping has not been observed.

COMMENTS

Inasmuch as the known facts regarding the etiology, pathophysiology and clinical course of Parkinson's disease have been recorded elsewhere by numerous authors, it seems unnecessary to restate the old story here. Suffice it to say that the abnormal neural mechanism that produces tremor and rigidity is not known and that all the explanations for these features of the disease must as yet be considered wholly philosophical. It is well known, however, that the disease advances or at least the disability resulting from tremor and rigidity is usually progressive and that medicinal therapy has not been very effective. For these reasons and others, it has been entirely proper to explore the possibilities of surgical therapy for those grossly incapacitated or in some instances for those who have completely withdrawn from society because of the unsightliness of their affliction. Certainly any patient with parkinsonism who is capable of continuing with his occupation, although somewhat handicapped, should be encouraged to do so and not to be imbued with the notion that an operation will restore him to normal.

There is a group of patients with parkinsonism who are relatively young but some have no gainful occupation principally because they are unable to obtain employment. Often after an unsuccessful struggle for a livelihood they are eventually incarcerated in a municipal institution. Here because of physical inactivity they rather rapidly become worse, especially as regards rigidity. Others are compelled to give up their jobs because of the incompatibility of the gross tremor and the technical skill required of them in their work. Still others, particularly young women, seclude themselves in their homes, unwilling to undergo the embarrassment attending average social activity. Among all of these there are some who may be restored to usefulness. The patients selected for operation should be under fifty years of age and not badly deteriorated physically. Tremor should be the outstanding feature of the disease and this for the most part limited to the extremities of one side. The patient and his family should have a complete understanding concerning the operation and what the outcome, barring complications, will be. They should be appraised of the future course of the disease and that surgical efforts will not, so far as is known, favorably influence the slow, down-hill course of the disorder. In short, the surgeon is operating for tremor and if, after careful assessment of all factors, it is thought that by abolishing the tremor the patient's life would be happier and the possibility of restoring him to gainful employment seems great, the operation should be carried out.

As stated, the average patient who has been submitted to section of the fibers of

the anterior limb of the internal capsule is able to walk, but with a mild limp, within two to two and a half weeks after operation. In some there is still a detectible residual slight limp after several months, however, this is scarcely noticeable and certainly not incapacitating. Gross purposeful movements of the paretic upper extremity return within ten days to two weeks after operation and during the ensuing month finer finger and hand movements are acquired. In fact, greater facility in performing skilled acts is often evident as contrasted with the pre-operative function. Frequently the patient will state that the affected hand is "normal," however by tests a mild dyspraxia can be demonstrated. If the tremor is abolished as described and a very slight hemiparesis endures, the possibility of a recurrence of the tremor is remote. Under unusual emotional stress transitory bursts of tremor have been observed in some, but these spells have been of short duration. Even though a very satisfactory result as regards tremor has been attained, in some patients the rigidity component while favorably modified is seldom completely abolished. If the proper selection for operation is made, namely, patients with gross alternating tremor limited for the most part to the extremities of one side, then rigidity is not a prominent feature. Marked rigidity and gross tremors do not co-exist. Recent experiences seem to indicate that rigidity may be favorably modified by an additional maneuver while performing the capsular fiber section; however, this of necessity will be subjected to further observations before conclusions are reached. It has been thought by several members of our staff that the so-called masked facies so characteristic of parkinsonism is less evident after operation. Possibly so, but certainly the blank, expressionless facies is not abolished. The same may be said of the greasy, sweaty skin. In many, it seems that this feature is less in evidence, however, no estimations have been made with precision. There is

no doubt that the general appearance of the patients on whom successful abolition of tremor has been accomplished is markedly improved. They stand more erect, walk with a "positive" stride and will smile under appropriate stimulus.

Of the twenty-seven patients with parkinsonism submitted to operation, fifteen had the procedure performed as herein described. Nine of the fifteen had as the outstanding manifestations of the disease gross alternating tremor limited for the most part to the extremities of one side. The tremor was abolished by operating in six, absent for the most part in one and diminished for six weeks to two months in two patients. The remaining six patients had involvement of all extremities, about equal bilaterally. In three of these the rigidity component was the principal feature, in fact they were designated as "frozen." All three of these died as a result of the operation: one on the fourth, one on the eighth and one on the eleventh postoperative day. Autopsies were carried out on two, however, the examinations failed to disclose any obvious cause for the deaths. The other three patients of the group of six had gross bilateral tremors, about equal on the two sides. Although unilateral operations produced a cessation of tremor on one side and this without paralysis, it seems questionable whether or not the situation as a whole has been improved in any of them.

One patient in the group of fifteen, upon whom the capsular operation was since performed, has had four generalized convulsions during the past five years. These attacks have not incapacitated him since. He has been employed as a watch repairman for the past three years, has had no tremor and says his hand is "normal." An additional two patients have also reached the five-year period since operation without recurrence of tremor. Moreover, no tremor has appeared in the extremities of the "uninvolved" side in any of these three.

The capsular operation has not been carried out bilaterally by me but Hamby has had experiences with three patients with alternating tremors upon whom he performed section of the capsular fibers on both sides. From his personal communication it would seem that the results were not good and that this particular procedure should be carried out only on one side.

As yet the operation has not been sufficiently perfected for me to produce an enduring cessation of tremor in all cases; however, more recent results indicate that one should be able to abolish tremor in a high percentage of patients selected as described. Certainly the results are sufficiently good to warrant continued efforts.

TRIGEMINAL neuralgia or trigeminal tic douloureux is referred to by various other names, including major trigeminal neuralgia and trifacial neuralgia. It is characterized by brief attacks of excruciating pain in the face.

The brief excerpts in this issue have been taken from "Surgical Treatment of the Nervous System" edited by Frederic W. Bancroft and Cobb Pilcher (J. B. Lippincott Company).

Presidential Address

THE AMERICAN PROCTOLOGIC SOCIETY

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AS we come to the close of another year in the history of our society, it is natural and fitting that we should pause to consider how much we have accomplished of what we set out to do and the means to achieve those things we have left undone. In approaching such an appraisal it is tempting, particularly for one of my age, to become reminiscent or to dwell at length on the future. Let me assure you this is not my intention. Many years ago Sir William Osler advised that man's life span should be contained in three lock boxes, one labeled "The Past," the second, "The Present," the third, "The Future," and that only the one labeled "The Present" was ever to be opened, on the theory that if one concerns himself diligently with the present, the past and future will take care of themselves. It is this pattern, the present, the immediate present, which concerns us.

It is apparent that times have changed since the organization of our society in 1899. The placid days when we had time to work out our own salvation are gone. We find ourselves living in an era of racial, religious and political confusion and unrest, with recurring wars and rumors of war that threaten the annihilation of the peoples of the earth. Our own government tended to go leftist, repudiating many of the principles upon which our democracy was founded and instituting bureaucratic regimentation in many phases of public relations, including our own profession. Any conclusions concerning our standing as physicians or special groups of physicians must take into consideration the effect these changes have had and will continue to have on the practice of medicine.

One who has had the honor and distinction of being president of this society for a year must of necessity have been impressed with the problems which confront our organization, a fact no doubt which explains the time-honored custom of the president addressing the society at the opening of each annual meeting. With this thought in mind, I would like to submit some of the important questions that have thus impressed me as being vitally in need of immediate solution.

MEMBERSHIP

The American Proctologic Society was conceived by a group of able, conscientious physicians who set out to establish proctology as an organized specialty of medicine. Through the succeeding years since its founding, the tradition and influence of this group have reached out like the ripples on a pond where a pebble had been tossed, touching the professional life of every member of this society. During the war doctors had an opportunity to see and evaluate the work of trained proctologists. Military patients, too, became aware of the difference between trained and untrained men in this field, all of which helps to explain the present growing interest in our specialty.

The most valuable asset of any organization or society is its membership. The American Proctologic Society has been a conservative organization but to keep abreast of its growing need, we have seen fit from time to time to increase our membership. At present there are over two hundred active members, the largest enrollment in the history of our society. We have reason to be proud and to believe this is the beginning of an expansion which will see our total membership equal that of the other organized specialities. In this connection I would warn you of the danger of carelessness or indifference in the selection of prospective members. So far we have kept the faith of our forebears by zealously adhering to the requirements for membership as set forth in our constitution which provides for membership by application. As long as we continue to adhere to our constitution we may be assured of the future. However, I firmly believe that we have reached a period in our growth when membership by invitation might better serve our society and better maintain our high standard of membership.

RECOGNITION OF PROCTOLOGY

This year marks the attainment of one of the most cherished ambitions of this society—recognition by the Council on Medical Education and Hospitals of the American Medical Association of proctology as an organized specialty of medicine. In a communication received by the chairman of our educational committee. Dr. Louis A. Buie, from Dr. B. R. Kirklin, the secretary-treasurer of the Advisory Board for Medical Specialties, Dr. Kirklin says:

"This is to notify you officially that the Advisory Board of Medical Specialties at its annual meeting held in Chicago on February 9, 1947, recommends to the American Board of Surgery that those desiring certification in Proctology *only* be certified in that field without having to take the entire examination in General Surgery, but, that those who wish certification in Proctology and Colon Surgery should continue to take the examination in Abdominal Surgery."

Dr. Buie also stated, "This is not all that we asked for, but I believe that it indicates that we are making some progress."

In an evolutionary growth such as we are making perhaps it is just as well we do move slowly, with time to adjust ourselves to the drastic changes incident to full recognition and the establishment of our own board, which are our ultimate aims. Thus, it will be seen we have by no means reached the top of the ladder. There are many and difficult problems to be solved and no doubt some tough sledding ahead before we reach this goal.

In the past there has been a fundamental requirement that every Fellow of the American Proctologic Society have good training in general surgery and proctology while no stipulation has been made as to the length of time required for this training or the conditions under which it was obtained. The caliber of the men who have composed our membership, the quality of their work and the exemplary conduct of our society have given our organization a prestige that has enabled us to press for and gain the recognition our specialty deserves. However, in the future, Fellowship in the American Proctologic Society must

be restricted to those men who are qualified to meet the newer requirements this recognition automatically demands.

Associate membership should be limited strictly to ethical practitioners, only those considered who are truly interested in proctology and who have sufficient training to be eligible to Fellowship when the time comes, that is, to men of ambition, with something to give to the society. We should not be content with a large part of our membership consisting of those satisfied to remain as associates or those seeking associate membership only for self-aggrandizement. Our society must continue to be interested in quality, not quantity.

RESIDENCIES

It is our responsibility to provide the facilities for the training required by the American Board of Surgery, namely, approved residencies. At first glance this would seem to be a simple matter. In reality it is one of the most difficult tasks that has ever confronted this society. First, there are not enough hospitals of approved standing to furnish sufficient residencies to meet the demand. What few residencies were available have been taken over by returning servicemen with priority based on previous service in said hospitals. Second, most of our hospitals, in an honest effort to cooperate in this emergency, have taken on more residents than they have work for and this has resulted in complaints from the residents that they are idle and not getting the experience they need. Third, with the exception of a few large institutions, the majority of hospitals state that they have not sufficient proctologic work to justify a resident in proctology. Fourth, another serious obstacle in the way of securing residencies is the attitude of the general surgeon toward proctology. The chief of the surgical staff of the hospitals which we must contact is usually a general surgeon, whose attitude only too frequently is that he is qualified to do all the necessary proctology. Therefore, there is no need for a specialist or a resident in

this field in his hospital. However, by proving to the general surgeon that we can do better proctology than he is doing, through better preparation, we have gained some recognition and cooperation from this group, as exemplified in the endorsement of proctology by the American Board of Surgery. This recognition by the surgical chiefs and surgeons who compose the surgery staff is a necessary step in obtaining the training facilities we need and constitutes an added responsibility for this society to continue to make good. Furthermore, many of the hospitals that we are asking to create new residencies in proctology have no well organized proctologic service. This is our opportunity. It is up to us to raise the standards of proctology in these hospitals or whenever the opportunity presents itself and by so doing create a demand for residencies.

A campaign should be launched to create dispensaries and, later, proctologic services in non-recognized hospitals located in cities and towns where Fellows of the American Proctologic Society are available to take over the responsibility of organizing and training interns and residents. I know of three hospitals which previously had no certified residencies where this was done by other specialties. Later, these services were approved and certified by the examining board of the Council on Medical Education and Hospitals of the American Medical Association. If other specialties can succeed in this manner, so can we. This is a gigantic task to be taken seriously by those who are assigned this work. I would urge that the committee fostering these residencies in proctology continue its contact with the Council on Medical Education and Hospitals with greater activity for it is not fair for us to require training with no available facilities to provide this training. I further would urge each member of the American Proctologic Society to dedicate himself to the task of helping in this crucial matter, that he consider himself a committee of one to spread the propaganda of good proctology in his state and local medical societies,

in his hospital staff society and among the interns, residents, nurses and his private patients for there is nothing as effective as united effort.

PROCTOLOGIC JOURNAL

On numerous occasions it has been proposed that this society publish a proctologic journal but nothing has ever been done about it. Formerly, these proposals were made when the need for a journal was not as apparent as it is today, when our society was small and the expense of such an undertaking did not seem in keeping with our financial standing. However, the most likely explanation is that societies, like individuals, hesitate to depart from established custom. If we are to keep in step with the acceleration of our day, it is time we seriously consider the advantages of a monthly, bi-monthly or quarterly proctologic journal instead of being satisfied with the outmoded publication of our yearly transactions.

First, we are a national organization. We encourage the formation of city, group and sectional societies, yet we have no cohesive intercommunication between these groups and the parent organization, the American Proctologic Society. Political, labor, church and other societies have demonstrated the value and need of united effort. A journal would provide this missing link in our national society.

Secondly, our membership meets once a year with no contact between the meetings except occasional terse announcements from our secretary. At each executive meeting vital measures concerning the policies of our society, about which our membership has had little or no previous knowledge, are presented and voted on. If we had a journal, these questions could be publicized, giving each member time to formulate a just opinion. This would minimize confusion, lessen the chance of mistakes and promote better understanding which makes for a better society.

Third, for those who attend our yearly meetings the material in our transactions is dead copy—a reference book only. Papers of value are being read before groups and sectional societies through the year that should be available to our membership at large. For example, I had the pleasure of attending the March meeting of the Philadelphia Proctologic Society. Three excellent papers and a review of the literature for the previous month were presented. Each of the papers read were of national society caliber and could have been accessible to all of our members if we had a journal. Yet I dare say only those present will profit by what was presented.

Fourth, our annual program of necessity is limited in the number of papers to be presented. This excludes many who would like to appear on the program and many who should appear. A journal would remedy this difficulty by providing a means for surplus new papers to reach the membership and it would encourage the new members, with little experience in writing papers, to develop this art.

Fifth, our honorary members, composed of men of outstanding proctologic knowledge, could be invited to contribute papers to appear in our journal. This would not only be another source of valuable material but would provide contact with a group of our society which has been neglected in the past.

Sixth, publication of announcements, obituary notices and open letters addressed to officers of the society, chairmen of committees or individual members on professional, political, legislative or personal problems, would make for a better informed membership.

Seventh and finally, the establishment of a Journal of Proctology is imperative in order that the aims and importance of the American Proctologic Society may receive full recognition. Had we possessed a journal such as is proposed during the past ten years, our struggle to place proctology in its rightful place with the other recognized specialties of medicine would have been accomplished in less time and with more decision than has been the case.

Having stated but a few of the arguments for a journal, and I dare say each member present could add to this list, the next consideration is the expense involved in such an undertaking. Last year our transactions cost this society $4,800.00.

An estimate from two reputable publishing houses on 300 monthly copies of a journal of one-hundred pages, paper and type to be the same as used in the average state medical journal, was $4.00 per page, or $400.00 a month, or $4,800.00 a year. Some of this cost would be absorbed by revenue from advertising. The printing bill, labor and postage incident to the reports and announcements sent out by our secretary is an item that could be deducted from this cost as such material would appear in the journal. Thus, it will be seen that the actual cost of publishing a journal would not materially exceed what we are expending for our transactions. There will be additional costs, of course, but the advantages to be had will be well worth the added investment.

The far-reaching influence of a Journal of Proctology upon the future development and growth of the American Proctologic Society, both within its own organization and in its contacts with other branches of surgery, must be evident to all of you.

FINANCIAL STATUS

It is an established economic fact that the cost of operation increases inversely with the expansion of a business or organization. The truth of this maxim was brought home to the Council of the American Proctologic Society at its Miami meeting. Here our treasurer announced we could no longer operate our growing society on the present hand to mouth financial basis; some new source of revenue would have to be found to carry on. The Council's reaction to this statement explains the unpleasant news you will receive in the form of an increase in dues and initiation fees, but this in itself is not sufficient. A growing society needs reserve capital for new projects which are a part of any expanding program.

In discussing with our secretary ways and means to build up such a reserve fund he offered this pertinent suggestion—that we interest our members in providing for the American Proctologic Society in their wills. It occurred to us that since gifts are deductible from income tax, our past presidents might be interested in making a direct gift which would be available to the society for immediate use. Also the membership in general might be interested in some form of life insurance with the American Proctologic Society as the beneficiary. This is done frequently by individuals who desire to leave something to a church, lodge, fraternity or some other organization. For those of our members who might be interested in this plan ordinary life insurance, with the society as beneficiary, could be purchased on their lives to cover any amount they wished to designate for a nominal annual premium. Such a plan would give those members the assurance and satisfaction that at their death the society would receive a legacy from them that would help build the future financial stability of the American Proctologic Society.

The foregoing suggestions are made merely to show the way for a new, sound financial policy with a definite budget system, which I maintain is imperative if we are to go forward.

SUMMARY

In closing, I wish to pay tribute to the sincere, active Fellows of the American Proctologic Society whose prolonged pioneering efforts in the years past, since the inception of our society, have been responsible for the remarkable achievement in the proctologic progress that has been attained. Time does not permit giving mention of those Fellows. Suffice it to say their names will always be a source of inspiration to those who will carry on. Whether or not this progress shall be sus-

tained is solely dependent upon the future activity of our younger Fellows.

As president of the American Proctologic Society I want to thank the officers, members of the Council, those who have served on committees and our genial and efficient secretary and his secretary for their earnest consideration and cooperation during the past year in making, what at first seemed to me an insurmountable task, one of the most pleasant experiences of my professional career. I wish to express my appreciation to those of our members and guests who are contributing of their time and energy by appearing on our program.

Also I want to thank the wives and sweethearts of our members for their gracious presence which always has added so much to the success of our meeting.

Last but not least, I want to express my sincere thanks to the members of this society for the privilege and honor of serving as your president during this past year. As retiring president, I pledge my best effort to the administration of our president-elect, with a prayer that 1948 will be one of the most successful years in the history of the American Proctologic Society.

PROLAPSE (rectal) is a condition of abnormal downward mobility or laxity of mucosa membrane. Actual protrusion through the anal orifice is not essential, for the prolapse may involve the mucous membrane lining of the sigmoid of the rectum and be obvious only upon digital or endoscopic examination.

Case Reports

MULTIPLE MALIGNANT LESIONS OF THE COLON

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NUMEROUS cases of multiple primary, malignant lesions, have been reported in the literature. In an article published in 1945, Bacon[1] gave a complete summary of cases reported to that date. The case herein reported is considered of interest as this patient had a right colectomy for carcinoma ten years before presenting herself with a far advanced lesion of the sigmoid which required a segmental resection and hysterectomy. An end-to-end anastamosis was subsequently done with restoration of normal function.

CASE REPORT

This patient, a sixty-nine year old white female, entered the hospital October 28, 1945, with a primary complaint of progressive weakness, the onset of which was two or even three years prior. This complaint had become markedly worse the last six to eight months before admission. Three days before admission she noticed blood in her stools for the first time since her former admission ten years ago. She had noticed dark but not tarry stools. She gave a history of alternating diarrhea and constipation for several months, but there had been no nausea, vomiting or any symptoms of obstruction and she had been in no pain since her former operation.

She had had rheumatic fever and the usual childhood diseases. In November, 1935, the terminal ileum, cecum, ascending and proximal one-third of transverse colon were resected and an ileocolostomy made, using the Murphy button, for a fungating annular adenocarcinoma of the cecum, followed by an uneventful recovery.

Physical examination revealed a thin, pale, emaciated white female of sixty-nine. Her head, neck, lungs and thoracic cage were essentially normal. The heart beat was rapid (120) regular; no abnormal sounds were heard. The abdomen was protuberant with a midline scar; there was no rigidity or tenderness. There was a suggestion of a mass in the right lower quadrant with a definite mass in the left abdomen, giving the impression of a colon filled with feces. The extremities and neuro-muscular system were essentially normal.

Laboratory findings were as follows: Numerous specimens of urine showed occasional pus cells; red blood cells, 3,590,000; white blood cells, 11,800; hemoglobin, 11 Gm. 65 per cent; differential and Schilling counts normal; clotting time three and one-half minutes; urea nitrogen 14 mg., occult blood in feces.

At the request of the medical section, the patient was seen by the author on October 30, 1945.

Proctoscopic examination revealed combined hemorrhoids grade 1 and no evidence of bleeding from any lesion at the ano rectum. The rectal mucosa and valves appeared normal. At 20 cm. two discreet adenomas were noted about 2 mm. in diameter which were removed for biopsy. The bowel was fixed and angulated at 24 cm., a large amount of blood-tinged mucus was aspirated, but the lumen could not be scoped with a small diameter scope because of its fixation.

X-ray examination on October 31st was reported as follows: "Injection of barium causes considerable distention of rectal ampulla and distal sigmoid. Complete obstruction is encountered in the region of the right lower quadrant and there is evidence of colonic delay above this point. *Conclusion:* Obstructive lesion of distal sigmoid probably malignant."

The patient was prepared for operation but declined, believing she was unable to stand a

FIG. 1. Photomicrograph of lesion showing microscopic appearance of lesion.

second operation. However, she then consented and was operated November 8, 1945, under spinal and pentothal sodium anesthesia. The abdomen was opened through a midline incision. Upon opening the abdomen, exploration revealed a large mass in the pelvis. The liver was palpated and found to be free of any evidence of metastasis, and no nodes along the course of the great vessels could be palpated. It was found on further exploration of the mass that the uterus and the sigmoid were adherent with an extension of the malignant lesion into the uterus. It could not be separated without breaking into the septic mass, and for this reason it was decided to do a hysterectomy and resection of the sigmoid as one unit. This was done by first dividing the round ligaments and Fallopian tubes bilaterally, which were in turn individually ligated with No. 1 chromic catgut suture. The dissection was carried around posteriorly between the peritoneal folds. The uterine arteries were identified and tied with suture ligatures of No. 1 chromic catgut. The uterus was then amputated, the cervical os was cauterized with tincture of iodine, and the cervical stump closed with interrupted chromic catgut sutures. The uterus was thus freed from its cervical and ligamentous attachments, but adherent to the colon. The bowel was then mobilized by freeing the peritoneal fold laterally, then medially and ligating the sigmoidal arteries and veins with No. 1 chromic catgut. The bowel was divided proximally above the mass and divided distally below the mass by use of the DeMartell clamp. A colostomy was contemplated with the proximal portion of the descending colon, but it was decided that the rectum could be mobilized. This was done by freeing attachments laterally

and its loose fascial attachments posteriorly, and bringing up the rectal stump. The two spurs of the colon were then approximated with a Rankin clamp. Sulfanilamide powder was loosly sprinkled retroperitoneally and intraperitoneally. The pelvic floor was peritonealized and the abdomen was closed in layers, using plain catgut to peritoneum, interrupted chromic to fascia and dermal to skin. Steel wire stay sutures were used. The patient was then turned on her right side and a drain placed into the retrorectal space, up beneath the posterior peritoneum of the abdomen to drain the area of oozing blood due to the freeing of the rectum from the intraperitoneal route.

A report of the histologic examination of tissue removed is as follows: "One specimen consists of several lymph nodes and a mass of hemorrhagic tissue, which grossly does not appear remarkable. Also present is a loop of large bowel which measures 37 cm. in length. The bowel is greatly distended. On section, the distention is found to be due to a tremendous tumor mass, which measures 8 by 9 cm. It protrudes 3.5 cm. from the wall of the bowel. Also present is a corpus uteri, which measures 6.5 by 5.5 by 3.5 cm. It contains a subserosal leiomyoma. The uterine body itself is not too remarkable. Microscopic sections present a papillary adenocarcinoma of the large bowel. Sections through the uterine wall present evidence of infiltration and there is also marked macrophage activity. The macrophages are unusually prominent. Sections of one adjacent lymph node present early metastatic involvement."

Pathological diagnosis: Papillary adenocarcinoma of the large bowel with obstruction and extention to the uterus and adjacent lymph node.

The postoperative course not remarkable. A Miller-Abbott tube was passed into the small bowel. The clamp to the proximal stoma was opened on the third day. An indwelling catheter was removed on the fourth day. A normal stool was passed from colostomy on the fifth day following a small oil retention enema. On the sixth day the tube was removed and fluids were given by mouth. The posterior drain was completely removed on the eighth day. The patient developed a sudden severe upper abdominal pain on the eighteenth day which subsided after seventy-two hours, the cause of

which could not be determined. She was discharged on the thirty-second day, having remained in the hospital longer than necessary because no adequate nursing home could be found at this time. Her general condition was good and the colostomy was functioning well.

The patient was readmitted to the hospital from the clinic on March 30, 1946. Her colostomy was functioning well, she had gained weight and strength and appeared well nourished. Her physical findings were essentially negative. She was urged to continue with her colostomy, but she insisted upon a closure as she desired to return to her native Sweden. She was prepared for surgery in the usual manner.

She was operated upon April 17, 1946. The colostomy was closed with a black silk purse-string suture after the usual abdominal preparation. The abdomen was entered by excision of the old scar. The abdomen was remarkably free of adhesions. The blind rectum was freed from adhesions and mobilized. The two adjacent ends of the bowel were then caught up in a Rankin clamp and the ends of each cut off with a cautery. A closed anastomosis was then performed, using a posterior and an anterior No. oo chromic catgut suture in the procedure as described by Rankin. The anastomosis was reinforced posteriorly and anteriorly with interrupted black silk sutures. A long colon tube was passed up through the anastomosis. Sulfanilamide powder was sprinkled lightly in the abdominal cavity, raw surfaces were peritonialized and the abdomen closed. A small rubber drain was left in the abdomen at the operative site, plain catgut was used for the peritoneum, No. 1 interrupted chromic for the fascia, interrupted black silk for the skin, with a small rubber tube drain being placed to the fascia.

Postoperative convalescence was wholly uneventful. The patient passed gas through the rectal tube; no decompression was necessary. The tube was shortened every forty-eight hours and removed on the sixth postoperative day. The patient had two bowel movements the following day. All skin sutures removed by the tenth day. The wound healed without drainage; normal bowel movements occurred and the diet was begun on the sixteenth day. After leaving the hospital, the patient presumably went to Sweden and there has been no follow-up note since.

CONCLUSION

A case of a seventy year old white female, who ten years previously had a right colectomy for carcinoma of the cecum, She returned with a far advanced carcinoma of the sigmoid, involving the uterus. Hysterectomy and segmental resection of sigmoid *en masse* was performed. A delayed end-to-end anastomosis of the colon was followed with complete healing and normal function.

The pathologic picture of the tumor is that of a papillary adenocarcinoma of the colon, most generally encountered in the right colon rather than the scirrhous type usually seen in the left.

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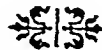
ISCHIO-ANAL DERMOID

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THIS case is of interest chiefly for two reasons: dermoids in an infralevater situation are, so far as I could determine, unreported, and although dermoids of the rectum are sparsely reported in the literature, they are almost exclusively found in the female. Dr. Arthur A. Landsman, in 1929, reported the only other case of such a lesion in the male; it was an intrapelvic dermoid pointing into the rectum.

CASE REPORT

Mr. J. A. McG., white, age forty-one, was admitted as an emergency patient to Hahnemann Hospital on November 7, 1946 with the diagnosis of ischio-anal abscess. A painful swelling in the left anterior perianal quadrant had developed approximately seven days before and the size and discomfort of the lesion had been increasing ever since. He gave a history of a similar abscess at the same site in 1939 which had been incised and unroofed. The surgeon had informed him then that the abscess was of an unusual type, being very deeply seated, and that further difficulties might be anticipated. His proctologic history was in all other respects unremarkable. Examination revealed a large, angrily inflamed, fluctuant abscess in the left perianal quadrant.

On the day after admission the patient was operated on under sacrocaudal intracaine anesthesia. The abscess was incised widely with the escape of a copious purulent discharge. After bevelling the margins of the incised abscess, further exploration of the tract in the portion proximal to the anus led to a deepseated pear shaped sac. This sac was dissected and removed in toto. It measured approximately 4 by 2 cm. When opened it was found to be filled with a large nest of hair, necrotic tissue mixed with feces and a fragment of a tooth measuring 0.6 cm. in its greatest diameter. Microscopic examination revealed the composition of the sac to be mostly connective tissue, with clefts lined by stratified squamous epithelium. Because the dissection had required a wound of excessive breadth, incision of the sphincter was deferred, a seton being placed through the internal sinus and the unopened portion of the tract.

The patient's postoperative course was highly satisfactory. By November 21st, thirteen days after operation, the wound had healed sufficiently so that the remainder of the tract could be incised and the seton removed. Two days later the patient was discharged. Subsequent healing was kindly and by November 30th was complete.

In this case the dermoid lay sufficiently close to the midline to lead the author to surmise that its origin was an invagination of skin during coalescence in the perineum. The mode of development of the communication with the anus is a matter of speculation.

In general dermoid cysts are benign. Malignant degeneration, although rare, does occur and may be carcinomatous or sarcomatous, with or without metastasis. If at all feasible, they should be excised in their entirety, not only as a safeguard against infrequent cancerous change, but also to put their harm to an end. In 1938 Dr. J. F. Saphir reported a case of true dermoid cyst of the anterior wall of the rectum which, of necessity, had to be treated conservatively although it occasioned much inconvenience to the patient. In his paper Dr. Saphir included an excellent review of the literature concerned with rectal dermoids, to which little has been added since.

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MESENTERIC THROMBOSIS FOLLOWING THE INJECTION TREATMENT OF HEMORRHOIDS

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IN a limited review of the literature I have found no mention of mesenteric thrombosis as a complication of injection therapy. Bacon[1] mentions hemorrhage and slough as the most common complications and ulceration, proctitis, stricture, abscess, fistula, liver abscess and pulmonary infarction as more remote complications.

Rankin, Bargin and Buie[2] mention four patients in whom disastrous consequences resulted from injection therapy; two of their own and two who were injected by others. These cases were all due to massive slough. Yaker[3] lists complications reported in the literature but makes no mention of mesenteric thrombosis. Morgan[4] in his article, "Castastrophies Following Hemorrhoid Injection," reports a case of cellutitis and slough of the entire anus and rectum.

Mabrey and Speare[5] add pelvic phlebitis and pyemia to the list. Rosser[6] adds formation of oil tumors or eleomas. None of the writers mention having experienced, observed or read of mesenteric thrombosis as a complication of this form of therapy.

The purpose of this case report is neither to discredit nor approve injection treatment of hemorrhoids. My policy has always been to limit injection treatment to the uncomplicated internal variety of medium size. If the patient states that his hemorrhoids protrude upon defecation and especially if they require manual reduction, I immediately suspect that they are not suitable for injection therapy.

CASE REPORT

The patient, Mrs. J. P. H., white female, age fifty-eight, was referred by her family physician on September 10, 1946. Her complaint at this time was swelling at the anus which was slightly painful, constant, not reducible and had been present for three days. The pain and swelling was not aggravated by evacuation. No bleeding had occurred nor had there been any protrusion on evacuation.

The past history revealed that she had experienced no severe illnesses, accidents or operations except a minor anorectal procedure performed in the office of a local proctologist four years previously. Her family history was irrelevant.

On examination the patient was well developed, well nourished, quite co-operative and apparently in no acute pain or distress. The abdomen was soft and not distended or tender. The liver and spleen were not enlarged and no glandular adenopathy was palpable. No masses were found. The perianal aspects presented a single external thrombotic hemorrhoid in the left lateral phase less than 2 cm. in diameter. On digital examination the external sphincter was slightly spastic.

Anoscopy revealed internal hemorrhoids of medium size in the left lateral, right anterior and right posterior phases. There was no ulceration.

Under proctosigmoidoscopy the bowel was visible for a distance of 15 to 20 cm. The mucosa was normal in color and no benign, malignant or ulcerative process was present.

Diagnosis: (1) External thrombotic hemorrhoid; (2) internal hemorrhoids, uncomplicated (medium size).

Ambulatory treatment was decided upon because it was thought that the patient's condition was suitable and also because she had an invalid husband from whom she was reluctant to be separated. Accordingly, the external thrombotic hemorrhoid was resected under local anesthesia on the initial visit. Two weeks later, after the external wound had completely healed, she was given her first injection. Five per cent phenol in Wesson oil was used and approximately 1 cc. was injected into the process in the right anterior phase. The usual care and technic advocated for injection

therapy was employed. One week later she reported for her second injection and a similar treatment was given in the left lateral phase.

The patient felt quite well until the evening of October 6, 1946, five days after the second injection when she began to have generalized abdominal cramping. Shortly thereafter she developed nausea and vomiting and her family physician was called. He did not consider her condition alarming until twenty-four hours later when she developed abdominal distention and tenderness in the left lower quadrant. At this time she was admitted to the hospital. There was no fever, the white blood count was 8,400 with 68 per cent polymorphonuclear neutrophils. The Wassermann was negative. A flat plate of the abdomen revealed several fluid levels in the upper left quadrant in the dilated small intestines.

A diagnosis of intestinal obstruction was made and the abdomen explored. Approximately 2 feet of the jejunum were found to be gangrenous due to thrombosis of its arterial blood vessel. This was resected and bowel continuity re-established by means of an end to end anastomosis. The postoperative course was uneventful.

SUMMARY

An apparently healthy individual with no history of previous operations or predisposing illnesses received two injections for uncomplicated internal hemorrhoids. The injections were given in the accepted fashion and no local symptoms developed. Five days after the second treatment infarction of a portion of the superior mesenteric artery occurred as demonstrated at the operating table.

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RUPTURE OF RECTOSIGMOID DURING SIGMOIDOSCOPY

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SIGMOIDOSCOPIC perforation of the bowel is not so uncommon as the few cases in the literature would seem to indicate. A mortality rate of 56 per cent has been reported in those patients not operated upon within one hour after rupture (Strömbeck). Any bowel, healthy or diseased, in any patient, conscious or unconscious, but especially past middle life, may be ruptured by any foreign body inserted into the rectum for any reason. The death of the patient in this case report appears to have been hastened by sigmoidoscopic rupture of the bowel because bleeding per rectum was the result of terminal and extensive cardiovascular disease. This serves to remind us of the ways in which such perforation might be avoided. In addition, the use of present day chemotherapeutic and antibiotic agents, as demonstrated in this case, might reduce the mortality in both those who are operated upon early, and those operated upon late or treated without laparotomy.

CASE REPORT

This is the story, in brief, of M. S. (Case 178728), a fifty-two year old white woman who was admitted on November 1, 1946 to the medical service of Dr. William Leaman at Philadelphia General Hospital for treatment of advanced hypertensive cardiovascular-renal disease. The prognosis was very poor.

On November 15, 1946 it was decided to take an x-ray examination of the chest, since, in addition to a pleural effusion, pulmonary infarction, hypertensive encephalopathy, oral hemorrhage and other signs of cardiorenal decompensation with edema, mild uremia, anemia and acidosis, this patient was also showing some evidence of a pneumonia and penicillin therapy was contemplated. As she was about to be lifted from her bed, it was noted that she was lying in a pool of blood which seemed to be coming from her vagina.

The gynecologic resident examined the patient and found a large clot in the vagina but no other pelvic pathologic symptoms. He felt that the bleeding was dysfunctional in type but that it might be part of her general cardiovascular disease or the result of a fundal carcinoma. He recommended dilatation and curettage if vitamin K, ergotrate and an icebag did not stop the bleeding.

These measures, in addition to plasma, failed to stop the hemorrhage and the fall in blood pressure. In the evening of the same day, therefore, the gynecologic resident performed a dilatation and curettage but found nothing to account for the bleeding. He placed a finger in the rectum and found a large clot of blood, whereupon he summoned the surgical resident who had done a few sigmoidoscopic examinations and the latter attempted to study the patient with the usual 25 cm. Montague sigmoidoscope which had been boiled but which was not sterile.

Being uncertain of his visible findings and having felt something "give" on introducing the sigmoidoscope, he asked the author to see the patient. When this reporter arrived in the operating room, the patient was on her back with her feet in stirrups and still under the effects of intravenous sodium pentothal anesthesia. The sigmoidoscope was in the rectosigmoid to a distance of about 14 cm. from the anus. Upon looking into the instrument one saw an elliptical opening, horizontally placed, on the right anterior wall about 1.5 cm. wide, through which had prolapsed approximately 2.5 cm. of glistening, pinkish white, serosa-covered small bowel with two fat tags attached to it. As the sigmoidoscope was withdrawn a little, a small quantity of pinkish, thin liquid gushed through the opening. Dark, thick blood could be seen coming down the lumen of an atonic sigmoid from above 14 cm. The mucosa was pale, edematous and wrinkled into folds. It seemed to invaginate itself into the sigmoidoscope. Above the anorectal line the rectal wall was heaped into thick, pale folds and small clots were visible, one of which was adherent to an

eroded area on the left just above the anorectal line and a small clot was in a vein beneath this area. No actual bleeding spot was encountered.

This patient had received no enemas either on the day of or the day previous to this examination.

Because of the patient's precarious cardiovascular-renal status and the presence of shock, immediate laparotomy was not done. It was hoped that the small bowel would become adherent to the ruptured sigmoid and that antibiotics would overcome infection. She was transferred to the surgical service of Doctor John Bower and placed in the Fowler position in an oxygen tent, given 500 cc. of whole blood; 200,000 units of penicillin intraperitoneally and 100,000 units intramuscularly every two hours, parenteral fluids and vitamin K (15 mg. daily); and rectal and upper intestinal Wangensteen drainage was instituted. The patient had been previously digitalized for her cardiac decompensation. She was also given Lugol's solution because a basal metabolic rate determined earlier had been plus 45 per cent.

On this schedule she developed no distention and had only slight rigidity and tenderness in the lower abdomen although no peristalsis was audible. Her temperature rose to 101.5°F. within the first few hours after the rupture occurred and then returned to practically normal until her demise. The pulse rate became accelerated and remained so. The blood pressure rose from 130/180 to 240/140 mm. of Mercury. The white count rose from 14,200 cells per cm. with 94 per cent polymorphonuclears two days before the rupture occurred, to 47,800 cells per cm. with 99 per cent polymorphonuclears the day following the accident. The red cell count was 3,500,000 per cm. with 9.5 Gm. hemoglobin. Serum chlorides approximated 515 mg. per cent, the blood urea nitrogen rose to 51 mg. per cent, the carbon dioxide combining power was approximately 41 mg. per cent and the urine output was diminished.

The patient continued to be weak, irrational and mentally dulled despite additional plasma, blood, fluids and penicillin.

Induration of the rectal wall was noted four days following rupture of the rectosigmoid.

The local surgical condition seemed to have responded to conservative treatment but the medical condition of the patient continued to become worse and she died on November 21, 1946, approximately six days after the perforation occurred.

The clinical causes of death were given as: (1) renal failure with uremia due to chronic glomerulonephritis, (2) perforated rectum, (3) pulmonary infarction, and (4) hypertensive cardiovascular disease.

At the surgical staff conference the patient's death was attributed to the patient's disease, to an error in diagnosis and to an error in technic.

The pathologic examination revealed the sigmoidal perforation to be closed by an adherent loop of ileum and an abscess, 4 cm. in diameter, was found in the pelvis. This yielded Bacillus coli, enterococci, Bacillus Welchii and Bacillus Friedländer on culture. Microscopically, the enteric vessels were thrombotic and the ileum was necrotic. Uncomplicated diverticuli were present.

Extensive vascular thromboses, embolism and infarction were encountered in the heart, lungs, spleen, liver and skin (which was also jaundiced), with the pelvic, left hepatic, portal, subclavian and cervical veins prominently involved. Hydrothorax, pericarditis, bronchopneumonia, adrenocortical lipoid depletion, patchy pancreatic necrosis, aortic and cerebral atherosclerosis and general passive congestion were additional findings. The heart was enlarged and contained a mural thrombus. The kidneys showed benign arteriolar nephrosclerosis. Traumatic cervicitis and endometritis were present. (The breasts were absent owing to surgical removal for tumors.)

The pathologic cause of death was given as: "Hypertensive cardiovascular-renal disease with myocardial failure; multiple venous thrombosis; perforation of the sigmoid with a pelvic abscess."

MALIGNANT MELANOMA (MELANOSARCOMA)

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THIS case is reported for four distinct reasons: (1) Because of the rarity of the condition; (2) as a plea for proper and painstaking examination which unfortunately is not always done; (3) to submit to microscopic examination every piece of tissue no matter how small or insignificant it may appear; and (4) not to be a proctologic agnostic who says that proctology consists of piles and nothing else.

Primary melanoma of the rectum is such an extremely rare condition that each authentic case report adds to the sum total of our knowledge of the disease. An important observation is the fact that when more than one case is seen, the diagnosis is made sooner because of one's previous experience.

The name melanoma is derived from its color which is a cross between violet and blue and is due to the deposit of melanin which is essentially of metabolic origin.

Ewing defines a melanoma as a pigmentiferous tumor arising from a specific mesoblastic cell, the chromatophore, and possibly also from epithelial cells which have been modified by pigment production. His view is that these tumors have their origin in the mesoderm yet histologically they appear as epithelium that has taken on a pigmentary function. He further states that Virchow recognized both a sarcomatous and a carcinomatous melanoma, the former exhibiting a diffuse structure of spindle cells, the latter an alveolar structure; Ewing employed the term melanoma for the entire group.

As to their origin, different views are held by different men but it has become the consensus of opinion and pathologists at present are leaning toward the view that all melanotic tumors have their origin in the nerve endings such as the tactile corpuscles. They are exceedingly malignant, grow more rapidly than carcinoma and meta-stasize to any organ of the body, including heart and spleen, by way of the lymphatics and blood stream. Secondary rectal invasion is not infrequent, owing to metastases from the original growth in other pelvic organs.

The description of melanoma in man was made in 1806 by Laennec. However, melanoma of the rectum was first described in 1857 by Moore. Paneth, in 1883, gave the first complete case record of a melanotic tumor of the rectum. However, the first comprehensive paper on the subject was written by Chalier and Bonnet in 1912, who, in addition to one personal case, collected and reviewed from the literature sixty-four reports of tumors of the melanotic variety and estimated that they represented 66 per cent of all rectal sarcomas and that they formed from 2 to 3 per cent of all melanotic growths in the body.

In 1936, Linder and Wood reviewed the literature and found seventy-nine authenticated cases. Weeks reports one case of large spindle-cell sarcoma and states that a review of the literature revealed one-hundred cases of sarcoma of the rectum. He further says that in 200 cases of malignant disease of the rectum, carcinoma is found approximately 199 times and sarcoma is found once. Of the rectal sarcomas the melanotic type appears twice as frequently as other varieties. This statement is well borne out by Kallet and Saltzstein who reported seven cases of malignant disease of the rectum, three of which were melanomas and one case each of myosarcoma and lymphosarcoma.

Allen, in the transactions of the American Proctologic Society of 1931, reported a case of melanotic carcinoma of the anorectal region which, at first sight, somewhat resembled a thrombotic hemorrhoid, from which these tumors must be differentiated

because melanoma of the rectum always occurs low down, either in contact with the epidermal portion of the anal canal or close to it.

CASE REPORT

Mrs. M. C., a white female, born in Russia, eighty years of age, was referred to me October 16, 1941. Two brothers had died of carcinoma of the stomach(?). Otherwise her previous history was negative.

She complained of pain and bleeding which had increased so that she decided "to do something about her bleeding piles," as she put it. She accordingly consulted a physician who removed her piles in his office by the fractional method. For awhile she was fairly comfortable but soon her "piles" came back and the bleeding and pain were worse than before.

Upon examination there was a bilateral mass originating at the anal verge. This mass practically occluded the anal canal excepting the right anterior quadrant. The left half was violaceous in color and semihard to the touch, while the right half was hard and had a crater-like depression in the center. Digital examination showed the sphincter to be rather tonic and another mass, hard to the touch, 1 inch long by ½ inch wide was felt in the left anterior quadrant.

The 10 inch sigmoidoscope was passed easily full length; the rectum and sigmoid were empty; the mucous membrane was normal in appearance; no gross pathologic condition was observed.

The barium colon enema and gastrointestinal series were negative. (These were done elsewhere.)

Having seen one other case, I suspected melanoma. She was therefore sent to the hospital for biopsy which was done under local anesthesia. Specimens from various parts of the tumor were submitted to the pathologist whose report was as follows:

Sections show hyperplastic tumor tissue consisting of large polyhedral and spindle-shaped cells with cyanotic nuclei and considerable variation in cell size and shape. A few multinucleated cells are also seen. Other cells have vesicular nuclei with distinct nuclear structure. Most of the cells contain melanotic pigment. There is very little stroma of fibrous tissue.

Diagnosis: Malignant melanoma.

MUCINOUS CARCINOMA ASSOCIATED WITH FISTULAS OF LONG-STANDING*

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THE occurrence of anal fistulas in intimate association with cancer usually poses the question of their etiologic relationship. At times the fistula is clearly the result of the breakdown of the neoplasm. But is the malignancy ever a sequel to the fistula?

There has been considerable skepticism in this regard. Jones[12] believes that the fistula in such cases is always secondary to the growth. Ewing[7] agrees with Kraske, whom he quotes, that "there is no evidence to prove that cancer develops in tissues altered by . . . fistulae or cicatrices." Kaplan and Rubenfeld,[14] during fifteen years on the Radiation Therapy Service at Bellevue Hospital, failed to find a single case in which a fistula was listed as antecedent to carcinoma. In Smith's[28] experience, "a fistula so rarely degenerates into cancer that the occurrence deserves notice."

Nevertheless, perusal of the literature reveals records of at least fifty cases in which cancer presumably developed in previously benign fistulas[1,2,3,5,6,8,9,15,16,18,19,20,21,23,26,27] or in scars following operations for fistulas or abscesses.[6,13,17,22,26,27,31]

It is difficult, almost impossible, in any given case to make certain that the fistula antedated the cancer. No matter how innocent its appearance, there is always the possibility that histologically the fistula may be harboring neoplastic elements. Even a negative biopsy report may be misleading.[5,6] The presence of tuberculosis,[21,31] lues[30] or lymphogranuloma venereum[5,16] may obscure a co-existing carcinoma. In addition, cancer may be so incredibly slow-growing as not to become evident for years.

Exhaustive histologic studies by Lynch and Gross[18] and Fitchet,[8] in an effort to establish a causal relationship between fistulas and associated carcinoma, failed to give convincing evidence.

Though proof be unobtainable, where a

TABLE I
MALIGNANCY ASSOCIATED WITH FISTULAS OF MORE THAN TEN YEARS

Authors	Duration of Fistula, (years)	Type of Neoplasm
David and Loring[5]	10	
	13	
Ducassi and Smith[6]	17	Colloid
	16	Colloid
Fitchet[8]	20	Squamous
Gabriel[9]	37	Squamous
Lisa[16]	10	Adenocarcinoma and squamous
Lynch and Gross[18]	15	Colloid
Meland[20]	10	Squamous
Moon[22]	10	Adenocarcinoma
Rosser[26]	15	Adenocarcinoma
	15	Adenocarcinoma
	10	Colloid(?)
Yeomans[31]	12	
	11	Colloid

fistula has been present over an interval sufficiently long to rule out the pre-existence of even the slowest-growing cancer, it seems reasonable to assume that malignancy supervened and was perhaps induced by the chronic inflammatory process. Such an interval, of course, varies; but set arbitrarily at ten years, it should be more than sufficient to eliminate any doubtful cases.

Reviewing the fifty cases mentioned, in which fistulas were inferably forerunners of carcinoma, we find that only fourteen qual-

FIG. 1. Case I.

ify under the ten-year interval limitation. (Table I.) In addition to these three of our cases fall in this category.

CASE REPORTS

CASE I. E. L., a sixty-one year old negro widow, was admitted to the service of Dr. Marino December 21, 1933, complaining of a discharging swelling of the left buttock. She had had an intermittently discharging fistula ten years, but during the last year the discharge had become almost continuous and a progressively increasing swelling about the fistula had developed.

On examination, there was a foul, cauliflower, ulcerated, non-tender mass, 6 inches in diameter, immediately to the left of the anal orifice, pierced by numerous openings, discharging mucopurulent material. (Fig. 1.) Two of these sinuses led to the ischiorectal fossa. In the left groin there was an oval, firm mass, 2 inches long. The blood Wassermann was four plus, but the Frei test proved negative.

A biopsy specimen from the perianal mass showed mucinous carcinoma. Figures 2A, B and C have been arranged by Dr. deVeer, Proctologist to the Brooklyn Hospital, to show, fields of progressively increasing pressure from the excess of mucus secreted by the tumor cells.

The enlarged inguinal node was removed and a few days later the tumor proper. It
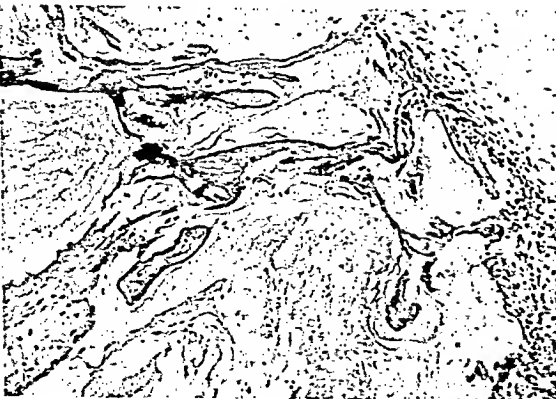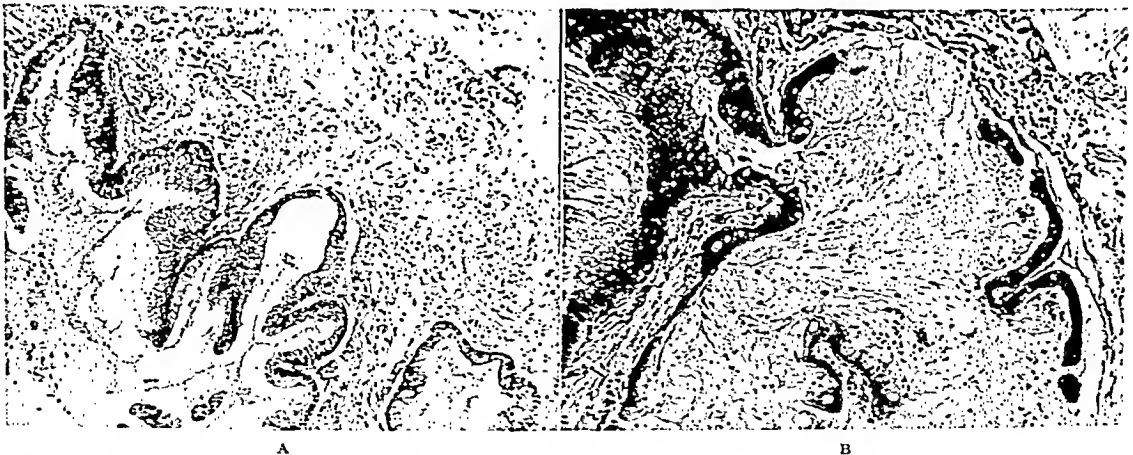
FIG. 2. Case I. A, the coarsely granular structure of the columnar epithelium is suggestive of rectal mucosa. Mucus is present in abundance within the cells and distends the gland lumina. B, cystic gland spaces in the tumor have ruptured so that mucus has been permitted to escape. The fragmentary remains of epithelium show shorter columnar cells with less secretory activity, evidently because of pressure atrophy. C, in this field abundant mucus has dissected connective tissue but no tumor cells are present. At this site the secretory tumor cells may have atrophied and disappeared, or the mucus may have been formed in some neighboring focus.

was found to be intimately attached to the lowermost part of the rectum. The wound filled in in two months. Six years later a recurrent growth was excised from the left groin and the following year another from the thigh just below the previous growth.

When I saw her in June, 1947, six years since the last operation and thirteen since the first, she was evidently in good health with no signs of recurrence.

CASE II. Mrs. A. D., a widow, aged sixty-four, was seen at her home in 1941 in consultation during an attack of pneumonia. Her daughter had noticed a perianal mass while inserting a rectal thermometer.

The patient related that she had had a fistula on the left side thirty years. During the last two years there had been an increasing, sticky discharge and a "hardening" of her left buttock.

The inner half of the buttock was infiltrated by a rigid mass, perforated by innumerable pinhead to pinpoint-size openings which presented droplets of glary, turbid fluid. One of these was found to be connected with a tract extending to the ischiorectal fossa.

During the remaining six years of her life she stubbornly refused to visit my office or to enter a hospital. I was kept informed, however, through her family physician. The growth inexorably progressed in size, ulceration and foul discharge. The patient gradually deteriorated and died of "pulmonary edema."

CASE III. M. C., a seventy-seven year old colored widow, was admitted to Dr. Marino's service March 21, 1945, complaining of fecal incontinence, perianal swelling and discharge.

FIG. 3. Case III.

She had had intermittently a fistula or abscess to the left of the anal orifice fifty-five years, but during the preceding few months the pain, swelling and discharge had progressively worsened and her stools had gradually decreased in caliber to pencil-thinness; incontinence developed.

She was orthopneic and dyspneic on slight exertion. Her heart was moderately enlarged and fibrillating. The liver border was two fingers-breadth below the costal margin. She showed poor renal function.

Locally, a rigid, friable mass displaced the medial half of her left buttock and extended anteriorly to the left labium, which was elephantiac, and to the opposite side of the anal orifice. (Fig. 3.) It was honeycombed with numerous sinuses draining a glary, mucopuru-

A   B
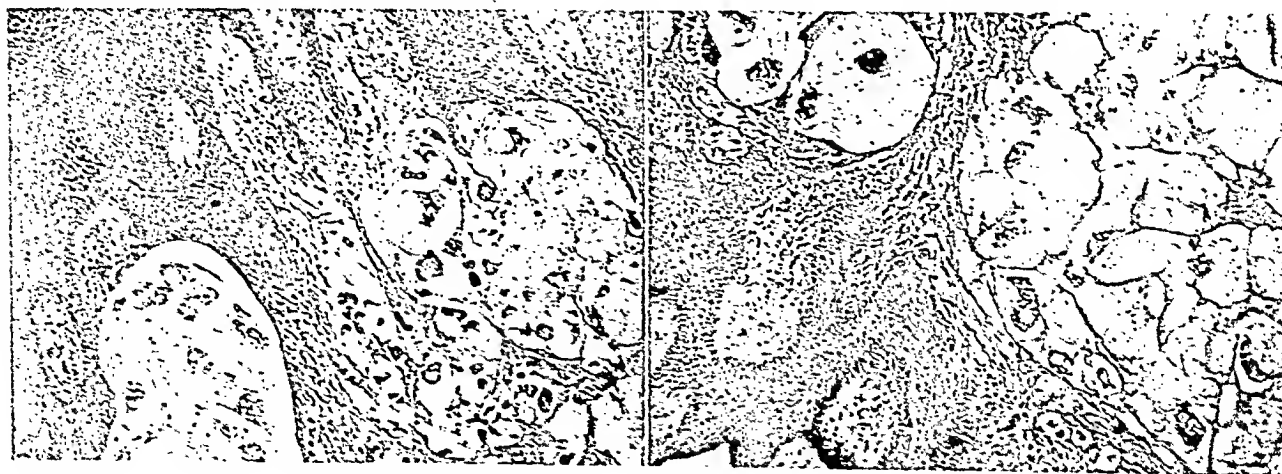
FIG. 4. Case III. A and B, the tumor structures consist largely of small glands and clusters of cells lying within masses of their own secretion. The mucus is seen dissecting its way up to and into the surface epidermis. These fields represent sites at which new fistulous openings were about to appear.

lent material. The involvement of the anus resulted in a stenosis which barely admitted the tip of the finger. In the left groin were small, firm, non-tender lymph nodes.

The blood Wassermann and Frei tests were negative. Because of absence of sensation in the tumor, a biopsy specimen was readily removed without the aid of an anesthetic. The pathologic diagnosis was mucinous carcinoma. (Figs. 4A and B.)

Because of the poor general condition of the patient, it was deemed inadvisable to risk surgery. However, she died two months later at another hospital of cardiac failure following a rectal resection.

COMMENT

These three cases bear a striking resemblance both in their histories and in the character of the growth. Recurrent abscesses and fistulas of many years' standing take on the aspect of an expanding, invasive neoplasm, begin to discharge an irritating mucous secretion in excessive quantities and ulcerate. These changes are gradual and progressive.

The question of the probable origin of this glandular type structure in the perianal region has intrigued many observers. It is believed that the answer is most likely to be found in the observation of Hermann[11] in regard to the presence of erratic glands, closed follicles and gland ducts connected with the anal crypts.

Besides exemplifying carcinogenic potentialities in old fistulas, these cases are of interest because mucinous or colloid carcinoma is very rarely seen in the anal region, particularly so in negroes. Of the fifty-one cases of anal carcinoma in the group reviewed by Buie and Brust[2] at the Mayo Clinic, only one was colloid in nature. Rosser[25] found only one of this type among thirteen anal malignancies in his experience. At the Brooklyn Hospital, Cases I and III were the only mucinous cancers among the twelve anal neoplasms in the past fifteen years.

It is especially remarkable that these two cases should have occurred in colored patients in a hospital predominantly white.

At Johns Hopkins Hospital carcinomas in Caucasians outnumber those in negroes four to one. Terrel[29] has seen only one anal cancer among negroes in Richmond which is one third colored. Gant,[10] with large negro clinics, never saw or heard of rectal carcinoma in a negro. Rosser[25] saw five cases and Kraker[15] one.

SUMMARY

Of the fifty cases of fistulas on record in which malignancy evidently supervened, probably not all truly represent carcinomatous transformations of previously benign fistulas. However, at least fourteen gave a history of a fistula of more than ten years' duration, long enough to rule out antecedent malignancy.

In addition, three of our cases are reported in which fistulas of ten, thirty and fifty-five years' duration, respectively, developed carcinomatous changes. These growths were all of the mucinous type, two of them in negroes.

CONCLUSIONS

Chronicity of a fistula should make us aware of the possibility of malignant change.

Whether or not the reported cases really represent cancer formation in previously benign fistulas, it is safest to eradicate fistulous tracts as early and as completely as possible.

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Papers of the Scientific Session

LEIOMYOMA WITHIN THE SUBSTANCE OF THE SPHINCTER

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SMOOTH muscle tumors of the gastrointestinal tract are of frequent occurrence in the upper segments but few have been reported involving the colon, fewer still the rectum and I can find no report of occurrence as distal as the

could find in the literature was a paper in Surgery, Gynecology and Obstetrics December, 1941, by Thedore Golden and Arthur Purdy Stout. They review the problem for the entire gastrointestinal tract and report one sigmoidal lesion which
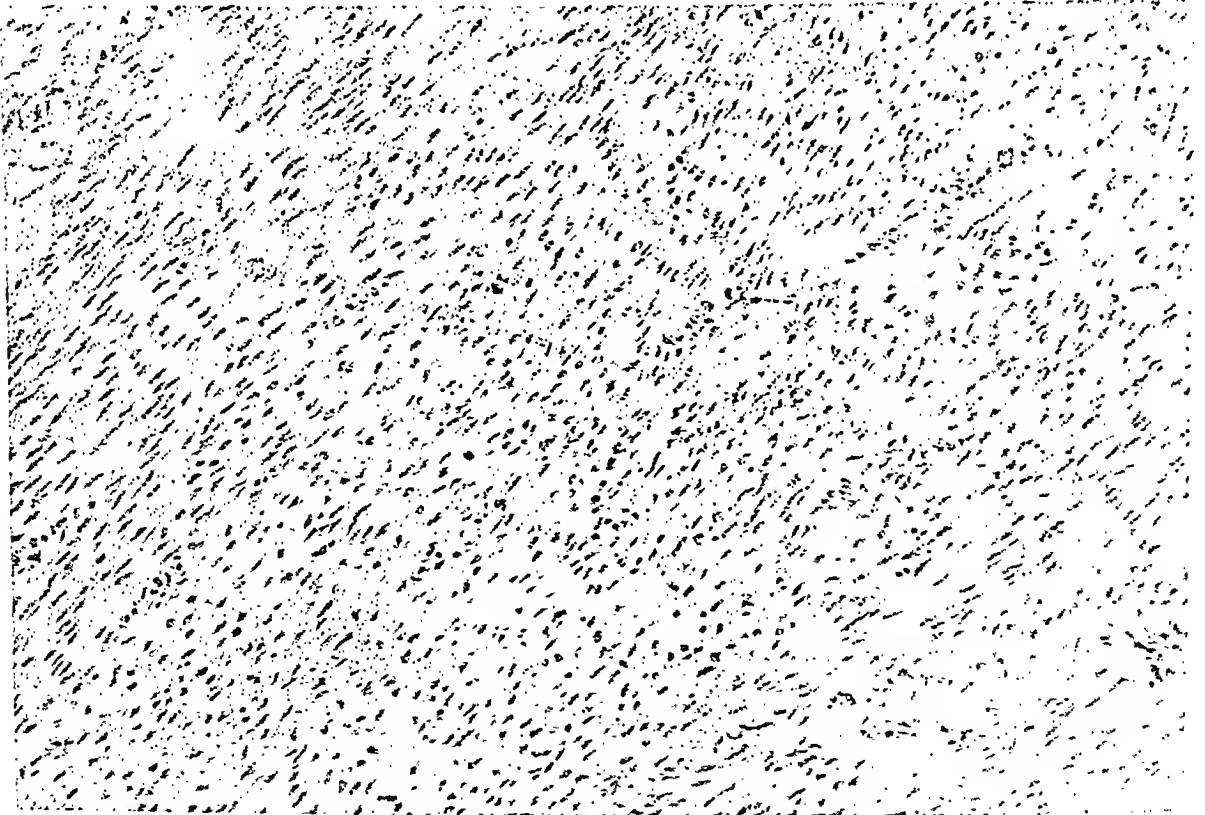
Fig. 1. Leiomyoma of anal sphincter.

lesion which I shall describe. Leiomyoma may be malignant or benign and is often sufficiently involved with fibrous elements to make the term fibromyoma more accurately descriptive.

The best resume of the problem that we

was malignant and one of the descending colon which was benign. Five rectal cases are reported: one of the lower anterior wall, one malignant tumor of the lower posterior wall, one not described, one pedunculated tumor of the mid-lateral wall and one

of the lower anterior wall removed, incidentally, in the course of perineal prostatectomy.

C. Allen Good, in Radiology of December, 1942, reports a large myoma of the distal portion of the transverse colon. This lesion was diagnosed by roentgen study, preoperatively.

William Lawrence Estes, in Annals of Surgery, Vol. XLIV, reports large myofibroma of the sigmoid. Diagnosis in this case was clouded by pathologists' suggestion of tuberculoma.

A bibliography of a report of a fibroma of the ileum by John W. Dewis, Boston Medical and Surgical Journal, October 18, 1906, includes four retrorectal tumors reported by four foreign writers.

H. H. Varner and J. L. Green Jr., in Southwest Medicine, 1935, Vol. 19, describe a small myoma of the sigmoid which was resected and primary anastomosis done.

CASE REPORT

R. P., a middle-aged female, was admitted to The Milwaukee County General Hospital on October 7, 1946 for treatment of purpura. The purpura responded satisfactorily to medical management and proctologist's consultation was requested on December 16, 1946 because of bleeding at stool and pain at the anus.

Examination revealed a firm, tender mass posterior to the anal canal. No fluctuation could be noted but surgery was advised for what was thought to be a small, chronic, postanal sinus or abscess. Upon opening the overlying skin it became obvious that the mass was a solid tumor. It was sharply demarcated and was easily dissected free from the normal sphincter muscle in which it lay. The tumor, about 3.5 cm. by 2 cm. in size, was of greyish red color and friable. The wound was left open to heal by third intention.

The pathologist's report was: "Leiomyoma, histologically benign."

It seems that the origin of this tumor must have been either the terminal portion of the longitudinal muscle or the fibers of the internal sphincter. Recheck examination on January 29, 1947 showed the wound to be well healed with no evidence of residual mass.

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PRIMARY POSTOPERATIVE HEMOSTATIC PROPHYLACTIC DRESSING IN ANORECTAL SURGERY

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THE demand for a prophylactic and hemostatic primary postoperative dressing in anorectal surgery has long been recognized. One that is less painful, non-irritating to tissue and not too bulky; one that is absorbable or easy to remove and simple in application; one that is adjustable to slight or tight, constant pressure to fit the need in the individual patient and one that will permit early expelling of gas and not interfere with catheterization or urination.

A new material in the form of oxidized cellulose gauze* used in conjunction with the sulfanomides has aided much in making such a dressing. The dressing consists of: (1) A thick strip of domestic or gauze to be used as a belt around the waist. (2) A large, perineal pad made of a piece of cotton ½ inch thick, 4 inches wide and 12 inches long, placed and folded in the center of a piece of gauze 28 inches long. This makes a pad 12 inches long with 8 inches of gauze on each end to be pinned to the bandage around the waist. (3) 5 Gm. of sulfathiazole or sulfanilamide powder. (4) One piece of 3 by 3 inch oxidized cellulose gauze. (5) One curved Kelly artery forceps to insert the oxidized cellulose gauze. (6) Two to four pieces of 4 by 4 inch sterile gauze pads. (7) Three large safety pins and (8) Two pieces of adhesive 2 inches wide and about 20 inches long.

APPLICATION OF DRESSING

The application of this dressing is most important. At the completion of the operation with the patient in a jack knife posture, 3 to 5 Gm. of sulfathiazole or sulfanilamide powder is dusted and rubbed

* Distributed by Parke Davis & Company under the trade name "oxycel.")

into the anal and rectal wounds with the gloved finger, care being taken that it is smoothly distributed and not left in piles to act later as foreign bodies in the wound.

Fold a piece of 3 by 3 inch oxidized cellulose gauze to match opposite corners thus making a right angle triangle. With a pair of curved Kelly artery forceps, grasp the folded gauze along a short side of the triangle so that the end of the forceps will be at a point of the long side. Insert the gauze with the forceps about 2 to 2½ inches into the anal canal so that it fits the slit-like shape of the anus and will come in contact with all the cut surfaces of the anus and lower rectum. This leaves half the gauze to be placed in or over the cut surfaces outside of the anus. Carefully remove the forceps so as not to displace the inserted oxidized cellulose gauze.

Place two to four pieces of 4 by 4 sterile gauze pads over the oxidized cellulose pack to fill in the space between the buttocks. Change the posture of the patient from a jack knife position to a ventral prone posture by flattening the table. Tie or pin the gauze band around the waist. Place the perineal pad over the sterile gauze pads and pin one end of the gauze, in the back, to the band around the waist. The patient is raised slightly to one side and the other end of the perineal pad brought forward, pulled tightly and fixed securely by pinning it in front to the bandage around the waist so as to make an upward and forward pressure on the anal dressing.

The dressing is completed by applying two strips of adhesive, 2 inches wide and about 20 inches long, strapped across the buttocks over the perineal pad. In people who have lost much weight or have relaxed redundant skin folds and poor muscular tone, the adhesive must extend well around

on the outer side of both hips to make constant pressure on the anal region.

This dressing is especially useful after hemorrhoidectomy for internal or combined hemorrhoids, anal, anorectal or rectal fistula, polyps, new growths or any other operation within the anus and lower part of rectum.

OFFICE DRESSING

As a dressing to be used in minor procedures in the office, such as excised thrombosed external hemorrhoids, skin, tabs, small anorectal polyps, anal warts, subcutaneous fistula, simple fissure and abscess, a strip of oxidized cellulose gauze placed or packed into the wound is usually sufficient dressing to control bleeding from the incised surface. A much safer and probably just as comfortable dressing is made if fixation and constant pressure is added. The shape and size of the oxidized cellulose gauze and the amount of pressure and fixation to be applied depends on the amount of bleeding and the size and shape of the wound to be dressed. As a rule the dressing can be removed in twenty-four hours without pain or bleeding.

VALUE AND USE OF OXIDIZED CELLULOSE GAUZE

Sufficient research and clinical use of oxidized cellulose gauze have proved that it is non-irritating to tissue and it has definite but limited hemostatic properties. Its value must be classed as only an adjunct to other methods of hemostasis.

Oxidized cellulose gauze applied directly to an oozing surface tends to adhere, a coagulum is formed and bleeding stops. When bleeding is brisk, the blood tends to push or wash off the oxidized gauze and prevent clotting. I have not found, as suggested by Rosser in the Southern Medical Journal, December, 1946, that small pieces of the material applied to each wound is sufficiently safe to be depended on as a hemostatic dressing. Certainly if bleeding is free, fixation of dressing and pressure should be added. If a spurting vessel is present, ligature is indicated and then the dressing applied.

Oxidized cellulose gauze when dry is pliable and easy to insert or apply to a bleeding surface. When the gauze becomes saturated with blood, it swells, turns dark brown and controls bleeding from the oozing surface. Within ten to twenty-four hours a film of gelatinous substance is formed around the gauze and the gauze turns dark brown and becomes friable or jelly-like depending on the degree of saturation with blood or fluid of the body tissue. This film prevents granulation from becoming involved into the mesh of the gauze, makes separation from tissue easy and, if pressure is not too tight, permits the early expelling of gas around the pack.

At the end of six to eight hours the strips of adhesive may be partly removed to see if blistering from the adhesive has occurred. If not, the dressing and packing is removed the next day. If the bleeding is sufficient to make complete saturation of the gauze, the gauze at the end of twenty-four hours is very dark brown in appearance and so friable that it may be easily pulled off in small pieces. However, in most cases, using gentle manipulation, the gauze can be separated from the tissue without discomfort to the patient. There is usually no bleeding in removing the gauze pack and the tissue is left dry, with little if any edema.

COMMENTS

After the dressing and pack is removed the wound is dusted with sulfonamide powder and two 4 by 4 sterile gauze pads are placed over the wound and fixed in place with short, narrow strips of adhesive. Hot fomentations are started in the afternoon of the second day.

The dressing should be applied quickly after the rubbing of the sulfonamide into the tissue and the mopping of the blood out of the wound to prevent further bleeding before the dressing is complete.

Hemostasis in this dressing is controlled first by the natural clotting of the blood second, by the hemostatic properties of the

oxidized cellulose gauze and third, by the constant pressure of the gauze against the bleeding surfaces to prevent displacement or expelling of the pack in vomiting, coughing, sneezing or exertion, giving time for clotting and the formation of thrombi in the open end of the vessels.

In the female, when cathterization or urination is necessary, the front portion of the pad may be unpinned and pulled backward out of the way leaving the rectal dressing secure and in tact. After the procedure is completed the pad is repinned in front.

I have used this dressing in more than 400 hospital cases. In no case has bleeding been sufficient to require a change or re-insertion of the packing.

When there is no evidence of bleeding on the bandage, pressure from the strapping of the buttocks may be released in from six to eight hours. When there is some evidence of bleeding on the dressing, pressure should be continued for twenty-four hours to give time for firm clotting in the vessels.

Beside the hemostatic effect of the oxidized cellulose gauze, the firm incompressible structure of the gauze makes it especially suitable for a dressing when pressure from the packing may be needed to control bleeding in the anus and lower part of the rectum, following hemorrhoidectomy, cryptectomy, fistulectomy, anorectal polyps, tumors, proctotomy and so forth.

There have been sufficient experimental investigation and clinical use of the oxidized cellulose gauze to prove its specific hemostatic properties. It has little value when applied to a spurting arterial vessel as the pressure from the bleeding vessel is sufficient to push off or wash away the clot. Therefore the spurting arterial vessels should be ligated and dependence should not be put on the hemostatic effect of the gauze to control this type of bleeding.

The perineal pad is made and wrapped in individual packages and sterilized along with other dressings in surgery. The chief objection that I have found to this dressing is that the adhesive strapping across the buttocks will occasionally cause blisters. This may be prevented by the early loosening (ten to twelve hours) or removal of the adhesive.

SUMMARY

1. A prophylactic hemostatic primary anorectal dressing has been described.

2. The method of application of this dressing is most important.

3. Sulfathiazole or sulfanilamide powder rubbed into the wound at the time of operation is the prophylactic portion of the dressing.

4. Oxidized cellulose gauze has specific hemostatic properties sufficient to control hemorrhage when applied directly to an oozing surface; when bleeding is free, definite fixation of dressing and constant pressure should be added. If a free spurter is present, a ligature should be applied.

5. Oxidized cellulose gauze saturated with blood turns dark brown and becomes friable. A thin, moist film is formed around the gauze within twenty-four hours, making removal easy without starting of bleeding. The tissue is left dry with little or no edema.

6. The thin waist film or coat of coagulum around the oxidized cellulose gauze pack permits early expelling of gas around the dressing.

7. An oxidized cellulose gauze pack if left in the tissue is absorbable.

DISCUSSION

HARRY W. CHRISTIANSON, (Minneapolis, Minn.): Dr. Pruitt has presented a very timely paper on the use of a relatively new postoperative hemostatic agent. He has described his postoperative dressings in very minute details.

We may not all agree with his technic of postoperative dressings but we can rest assured that his method accomplishes the end result.

I do not agree with Dr. Pruitt in the application of oxycel in every operative patient but we cannot deny the fact that this oxidized cellulose gauze or similar hemostatic agent is a very valuable adjunct to our armamentarium in preventing and treating postoperative hemorrhages.

Dr. Pruitt wisely admonishes against the use of oxycel when arterial bleeding is present.

Suturing then is absolutely indicated if the surgeon wishes to sleep that night.

We use oxidized cellulose gauze or similar preparations postoperatively, only when generalized venous oozing is present. This avoids the use of excessive suturing and hence reduces the postoperative pain as every suture is a potential producer of pain in the anoderm and perianal skin.

The destruction of polyps in the rectum or sigmoid by fulguration in my experience has never indicated the need of any hemostatic agent as fulguration is sufficient. However, if we fulgurate a large, sessile polypoid mass with or without malignant degeneration then oxycel is placed over the area and held in place with packs of gauze.

When the need for oxidized cellulose gauze is indicated, we usually insert this hemostatic agent in the rectum in the form of a cylindrical roll until 1 inch or less protrudes from the anus. The protruding exterior portion of this "oxycel" pack is then split longitudinally and reflected laterally to cover any raw surface external to the anal margin. Four by 4 inch gauze sponges are then packed in between the separated ends to fill in the space between the buttocks. This places the hemostatic agent against the raw surfaces after which an adhesive strap is applied tightly for pressure. Three to four hours postoperatively, the dressings are removed but the oxidized cellulose gauze is left intact. The oxycel is removed in forty-eight hours or, if deemed advisable, it is left in place and expelled later with the first bowel movement.

We do not recommend the routine application of sulfathiazole or sulfanilamide to anal and rectal wounds; however, in grossly infected patients its use may be indicated.

Before the advent of these hemostatic agents such as oxycel, thrombin topical and gelfoam we sometimes had to resort to large gauze packs to control postoperative bleeding. The removal of these packs was very painful and frequently required the use of an anesthetic. Now in its stead we use one of the hemostatic agents, preferably "oxycel." The removal of these packs is painless; in fact, it is usually unnecessary to remove them. Dr. Buie would say, "why worry, the current is always to the south."

In conclusion, I want to commend Dr. Pruitt on his usual fine presentation of a paper and on his observations on the effect of a new and valuable aid to postoperative hemostatis.

Dr. Jack Kerr (Dallas, Tex.): Oxycel gauze is the outgrowth of the search for agents that would carry the administration or the application of thrombin, thrombin, of course being a very powerful coagulant. This search for a material to be absorbable and yet non-irritating has been going on for some while, the thought perhaps being advanced many years ago, even by Cushing, when various experimental studies were carried on with fibrin itself. The first of these preparations was fibrin foam, which later included oxidized cellulose, both cotton and gauze, and the gelatin sponge and starch sponge.

As it is marketed—oxidized celluloses, both the cotton and the gauze—if thrombin is to be used topically with the application, alkalinization becomes necessary. This, of course, is done by the addition of a 1 per cent sodium bicarbonate solution to the gauze after which it becomes very friable.

In later studies, it was found that these oxidized cellulose preparations alone had certain hemostatic properties, perhaps not as good as thrombin itself, but definitely present. They are also described largely on the basis of a styptic action but apparently there is some chemical reaction between the cellulosic acid, which oxidized cellulose is, and the salts of hemoglobin.

Our experience with these preparations includes three: the oxidized gauze and cellulose and the gelatin sponge. Our personal preference for these three is the gelatin sponge soaked in thrombin. It is a little more easily applied and, of course, including the thrombin we believe that the hemostatic properties of this is superior to the oxidized cellulose.

I think Dr. Pruitt is eminently correct in emphasizing that these types of dressings are not hemostatic to the extent that they exclude the necessity for ligature of any definite bleeding that is occurring at the close of surgery. A generalized ooze, as Dr. Christianson mentioned, is a definite indication for these preparations; they help a great deal. I do believe some amount of pressure is best. It does decrease the amount of suturing that is necessary and, therefore, cuts down the amount of postoperative spasm and pain that results following anesthesia.

We do not believe that these preparations should ever be used to the exclusion of the ligature method as Dr. Pruitt mentioned.

I enjoyed Dr. Pruitt's paper very much.

RELATION OF FUNCTIONAL TO ORGANIC DISEASES OF THE ANUS, RECTUM AND SIGMOID COLON

HENRY C. SCHNEIDER, M.D.

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DURING the past five years there has been a notable impetus given to the work of Freud and his associates in their studies on the relationship of the psychic behavior of individuals toward disease. This has been largely due to the influence of the war years, during which time great opportunity was offered in the mass study of the great number of men who were selected or rejected for service in the armed forces. The strain of combat also yielded excellent opportunity for the psychiatric study of a large number of men under abnormal conditions. The fortunate part of the entire study was the fact that strenuous efforts were made in standardizing psychiatric diagnoses and evaluating them on a scientific scale.

It must be realized that here we are dealing with something intangible. Bold and often strange theories may be presented which can only be given consideration as such until improved physiologic methods are devised to aid in final evaluation of the mechanisms responsible for functional disease. In this presentation, it is not possible to discuss the details of this work as the ramifications are considerable and it is a field in which special training is necessary for good exposition of its use, value and correct approach. However, there are many occasions in common daily practice in which the knowledge of the principles of psychosomatic therapy can be used to advantage. I believe most of us have the feeling that when psychosomatic problems are discussed, the concept becomes one of exclusion of organic disease. If no organic disease is found, then it too often is considered a functional problem and the patient is labeled a neurotic. At this stage the condition is treated symptomatically without any further attempts to inquire into the background or offer any help to the patient from a treatment standpoint of his functional disease. The problem should be evaluated briefly and if found to be deeply seated, requiring much time and possibly specialized knowledge, then referral to a psychiatrist is advisable. Not only is it possible for both functional and organic disease to be present at the same time but its combination exists in practically all diseases of the anus, rectum and sigmoid in varying degree. It must follow then that we become interested in the *entire* problem. Weiss[1] stated, "Psychosomatic medicine does not mean that the soma should be studied less but the psyche should be studied more."

Most psychosomatic diseases are based on the existence of a vicious cycle consisting of psychic and somatic factors and, depending upon whether or not it is the psychic or somatic component that is cured, the cycle may be broken. Therefore, a psychoneurosis may run an acute course and recover spontaneously when the basic somatic disease disappears, providing this disease is the precipitating factor. It is also possible that some unrecognized organic disease may very often stimulate a neurosis or cause a neurotic condition.

I am sure that you have all made the observation that most patients visiting a proctologist, at least for the first visit, are usually frightened, nervous and anxious. This is especially true of the female patients and, therefore, great care must be taken to put the patient as much at ease as possible. The first meeting of a patient with a physician can be one of confidence, trust or dependence; or it can be one of distrust, caution or defiance. The psychiatrist

states that transference neuroses may immediately be founded at this meeting. For instance, in the background of the patient there may have been a childhood relation of distrust toward the parent. This distrust or anxiety may be transferred to the physician and a neurotic attitude develops. This fact is important when the referring physician states, "I have examined this patient thoroughly. I am sure she is a functional problem, but I am unable to convince her that she does not have organic disease. I want you to handle this problem."

A more detailed history is important in the study of functional proctologic problems. A general rule that might be followed in the selection of a neurotic is that they are usually overactive and oversensitive. They may object or even refuse to give details of their childhood or their relationship with their parents or close relatives. In these individuals it is important to deal more thoroughly with a short analysis of the problem as it is presented by the patient.

During the examination procedure draping, comfortable position and gentleness must be given its proper attention. A physician with a reputation of kindness and gentleness will always be greatly appreciated. During the examination it is well to continue conversation with the patient in order to divert him from fear of the examining procedure. One of the greatest anxieties of the majority of patients is that a malignancy is present. If following the examination no malignancy is suspected or found, it is important that the patient be told this fact immediately and in an assuring tone. This follows the psychotherapeutic principle that when neurotic symptoms are divorced from the fear of organic disease, cancer for example, it loses the force, whereupon the slogan "carry on in spite of symptoms" often helps the patient a great deal.

One must face a psychic problem when recommending hospitalization or surgical procedure. Many patients have a great fear of any type of operation no matter how minor and even the thought of being admitted to a hospital is a great shock to them. It is, therefore, important that this be done in a manner which does not impart a great impact. This can be done by appealing to common sense values. A word might be added as to the worth of a secretary or nurse who is courteous, polite and is taught these same principles.

Much has been written and attributed to the physiologic act of defecation. Psychiatrists are very prone to believe that many of the mental disorders in adults and children can be attributed to abnormal habits concerned with this function. Whether this premise is correct or how frequently it occurs is not the purpose of this presentation. However, many psychologic problems are discovered to be the source of diagnosis in children of fissure-in-ano or poor bowel habits who are often referred by a pediatrician. The organic pathologic condition is preceeded by poor bowel training in many instances and often a definite bowel complex is already present in these young children.

The matter of bowel function in children and its relation to the psyche can be explained as part of their normal development. The first year or two of a child's development is the oral stage and the following two years can be considered the anal stage. Here children find that they have control of the sphincter and there is pleasure in its realization. Instead of it being a natural function and consumated when desired, it is, at an early age, definitely molded to fit the needs of our mode of living by having a movement at approximately the same time each day in an unnatural receptacle and position. The child will give up the newly found control if a pleasurable state can be substituted such as love for the parent and the surroundings. This is usually sufficient compensation for the average child. Difficulty in bowel training arises very often because of the exaggeration of its importance by the parent or overaccentuation of the basic return by the child as compensation

for the giving up of the pleasurable act. If toilet training is begun too early in life, the child may feel punished and think he should never have a defecation. The bowel habit then becomes irregular, a laxative is given by the parent the child refuses the laxative because he knows what the laxative will accomplish and believes it is not the right thing to do. In adult life it is the extremely meticulous housewife type that shows a carry over of this condition. It often becomes a compulsive state which is very difficult to overcome. In the treatment of functional types of constipation and diarrhea the proctologist will often find emotional factors which are the basis of the dysfunction. This is true not only in adults but in children as well. The factors involved have been already referred to but it may be stated that fear and anxiety increase the secretory and motor functions of the colon producing diarrhea while depression and despondency decrease these functions and produce constipation. Von Bergmann and Lenz[8] have demonstrated a mass peristaltic movement which occurs one or twice daily, consisting of the sudden relaxation of a physiologic sphincter located near the distal portion of the transverse colon with a simultaneous contraction of the cecum and ascending colon. If this fails to occur, it can be due to either atonia because of the lack of propulsive force or blocked by spasm in the spastic type. With this in mind a proctologist can often recognize these simple principles and clear up a situation which is most important to the patient and the parent.

A little psychotherapy will go a long way in postoperative treatment which may produce a painful response. Talk with the patient in order to divert his mind from anxiety.

Mucous colitis or irritable colon is probably the most common proctologic ailment which is largely of a functional nature and can be greatly improved by psychotherapeutic treatment. Because of this relationship, the condition has received a great amount of attention by the psychosomatic

school. White, Cobb and Jones[2] regard mucous colitis as a bodily reaction rather than an organic disease. It is apparent that the autonomic nervous system plays an important rôle in the syndrome of irritable colon and that this dysfunction can be brought about by emotional factors. In general, it might be stated that stimulation of the parasympathetic system produces increased muscular tone and peristalsis with relaxation of the sphincters. Stimulation of the sympathetic system produces relaxation of muscular tone and diminishes peristalsis with spasm of the sphincters. It is possible to furnish evidence that there is active instability of the autonomic nervous system in persons with mucous colitis. This can be established by making use of various physical efficiency tests, among which there are the tests of Schneider[3] and Turner.[4]

There appears to be a definite type of patient who exhibit the intestinal findings of mucous colitis. They have been divided into three groups according to Alexander, Kahn and Adler: The first type, or the upper gastrointestinal type which exhibits cardiospasm and peptic ulcers, are usually active, independent, efficient and show overcompensation for inferiority. The second group, or lower gastrointestinal type, usually show symptoms of diarrhea and are definitely of the inferior type who are dependent, have a sense of guilt and a need for giving. The third type, also lower gastrointestinal usually showing constipation, are the vain individuals, having no sense of guilt or a need for giving and depend upon prestige for their receiving. The collection of these three groups with emotional makeup so expressed might give a clue to the proctologist in the recognition of an undetermined basic psyche. Jordon[5] has stated that we are accustomed to think of functional gastrointestinal diseases in two categories: (1) that which is psychosomatic, altogether or in part and (2) that which is somatic in origin. It is her opinion that symptoms of short duration are often on a psychogenic basis and in these instances

psychotherapy is completely effective. In the group that show symptoms of longer duration even though psyogenic in origin, it is necessary to treat the somatic condition. In the second group of patients she believes functional improvement is on a somatic basis.

A word concerning the radiologic evidence of mucous colitis might be important at this point. With the ordinary preparation used by most radiologists for a roentgenologic study of the colon, spasm and irritability are set up even in normal individuals by the laxative or enema used in the preparation. This will make the study valueless insofar as the diagnosis of mucous colitis. The ordinary preparation should then be considered as a search only for organic disease of the bowel. It becomes necessary to consider an additional study when one is looking for functional disturbances and in this instance x-ray should be performed without any preparation whatsoever.

In evaluation of the diagnosis of mucous colitis it is important that all tests be done first so that organic disease is fully eliminated before the diagnosis and treatment is given the patient. If the proctologist back-tracks later and asks for tests to rule out organic disease after having stated that only functional disease existed, all confidence toward the physician is lost by the patient. It should be explained that his symptoms are due to overexcitement of the bowel and that he should never use cathartics. A low residue diet and elimination of any irritating physical factors are important. The use of a synthetic atropine derivative is often helpful in the acute stages of the disorder. It is important to discover the cause of tension in each of these patients. If the correct insight is given, adjustment made in their activities and constant reassurance given, the emotional tension producing the attacks can be lessened.

The psychosomatic factors concerned in ulcerative colitis are only debatable insofar as the extent that the emotional back-

ground plays in the etiology and treatment of the disease. Murray and Sullivan[6] have stressed the importance of psychogenic factors in the etiology of ulcerative colitis and consider psychotherapy very important in its treatment. They believe that when the emotional conflict is solved and there is no longer this irritating factor, intestinal motility returns and the bowel can take care of any bacterial invaders so that the disease should promptly improve. The fear of the disease in itself, the fact that it occurs most frequently in young individuals who are often the sole means of family support will cause psychic factors which aggravate the patient's general condition and, in turn, the local disease. These factors, therefore, should be either eliminated or at least ameliorated. Bargen and Jordan,[7] in a symposium on the treatment of chronic ulcerative colitis read before this society in San Francisco last year, attested to the great importance of psychotherapy in the treatment of the disease. I believe that a practical view can be taken regardless of the actual etiology of the disease, whether it be organic or psychic. A certain varying degree of emotional back-ground can be found which has been present previous to the initial disease or immediately before a flare-up of the chronic form in almost every patient afflicted with the disease.

Pruritus ani also has its psychiatric elements. The real percentage of patients with pruritus who have a functional background cannot be stated. Regardless of this, I believe it is important for us to know that in any of the patients, even when it may be entirely of organic etiology, there is most often a varying proportion of mixed psychic disturbances and organic elements. Stokes[9] has cited the case of an elderly gentleman who refused treatment of his ringworm with the frank remark that the disease and its attendant scratching resulted in more pleasant sensation than sexual relations with his wife. This instance represents the masturbatory element that is associated in patients with

pruritus and shows the masochistic element inherent in the concomitant suffering and pain. These patients are usually overactive and oversensitive. Overactivity results in exhaustion and oversensitivity produces exaggerated emotional changes which increase the exhaustion. The fatigue does not stimulate the patient to the ordinary tired feeling but stimulates abnormal reflexes and causes lack of inhibitions. For example, increased sweating in an overactive patient with a definite fungus infection results in more optimal growth requirements and might explain a flare-up of fungus infection.

CONCLUSIONS

It has been the aim of this presentation to show briefly that psychic disturbances of varying degree are present in the majority of proctologic disorders. If should be recognized that since organic pathology may produce psychic disturbances and functional disease can produce organic disease it follows that they are usually present simultaneously. It is important that the psychic component be evaluated and, if necessary, treated in the light of our present concepts of psychotherapy.

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DISCUSSION

RICHARD H. APPEL (Indianapolis, Ind.): I find Dr. Schneider and his paper to be much like a troublesome conscience that has come back to plague about a personal weakness which is a dislike of the treatment of psychopathic patients. As a result, my efforts in the past have been directed chiefly towards a diagnostic end. So to a certain degree, I represent a negative side in this discussion. I am sure that one of the reasons which led me to enter the field of proctology was the idea that the pathologic conditions encountered were both visible and palpable and it is with this attitude that most of our competitors in the general surgical group begin and end. I have learned that this is not true, that often the task of separating somatic from the psychic presents one of the most difficult of diagnostic problems. It is here that our knowledge and judgment attain their highest value because we are often called upon to be the final judges in this question. This group of patients is the private preserve of the proctologist whether he likes it or not. There is no overlapping field for the two branches of proctology and psychiatry and often patients pass each other between the two offices. Particularly in adults, however, more are apt to arrive in proctologists' offices than to depart.

I find that I am more comfortable in the presence of the psychiatrist since the war than before. Perhaps that is because I saw more of them in the service, lived with them and may even have been somewhat inoculated with some of their theories but I do not believe so. I learned to respect them but found that V-J day cured more of their patients than any single therapy. I think they would be more grateful to us for proctologic therapy than for assimilation of their theory. I understand there is now considerable discussion among them as to the importance of organic dysfunction in contrast to those who are interested chiefly in functional disorders as a basis of the patient's trouble. Apparently there is no retreat from Freud but there is less emphasis. So our efforts, I believe, should be directed towards greater diligence in correcting minor defects. This is frequently

done at the instigation of the psychiatrist himself, however, with mixed results.

I do find myself looking more and more closely at the increased thickness of the rectal mucosa, very slight prolapse, increased sphincter spasm, etc., and mulling over the minor complaints.

W. A. H. SCHEFFLER (Camden, N. J.): Research to measure and make perceptible to our senses and control, forces which were not visible or perceptible before, lately has been making great strides—stratosphere, cosmic rays, radar and nuclear fission are just a few examples.

It behooves medicine to make the same progress in the evaluation of processes which are not directly perceivable and measurable but rather intangible. I think Dr. Schneider's paper has contributed in a most excellent manner to this problem with his study of the psychosomatic responses and their relation to the gastrointestinal tract and I wish to congratulate him.

The few minutes available for discussion will not permit me to go into many details of this paper. I am therefore trying not to repeat anything that has been said and just elaborate on a few points of this fascinating subject. Dr. Schneider mentioned the anal phase of the child, during which toilet training takes place. The psychiatric conception is that the infant has only *received* until this anal phase and the first thing he is expected to *give* or contribute for the benefit of the family is evacuation at the proper time and place. Instinctively, the child expects a reward for this, his first contribution to the family comfort, which seems of extreme importance to him. Love, fondling and praise mostly will be all that is expected by the infant. The adult who reverts back to this means of attaining compensation expects much more; understanding of his problems, patience even with his illogical reasoning and so on.

If infant toilet training is done too quickly, strictly or in a punitive manner, the impression may be created that the normal elimination process is dirty, immoral and something to be ashamed of. (The result might be fear, over-modesty and apprehension in later life under certain provocating stresses.)

If economically, socially, financially, professionally or sexually frustated or unsuccessful, the neurotic patient may revert back to the infant tendency to get a reward for his gift ability, the ability to give feces.

Neuroconstipation thus could sometimes be regarded as the desire *not* to give as a defense or an anxiety to offend somebody as the infant was taught in childhood that a bowel movement could be offensive. (These latter patients often cannot urinate or defecate in presence of others.) This may be a response to anxiety, resentment or guilt and may be associated with depression, neurasthenia or over-conscientiousness (the rigid type). (It also may lead to diminution of libido.)

If *acute* physical or mental trauma occurs in later life which the patient is unable to cope with, he likewise may regress to the childhood stage—which was more pleasurable and satisfactory—even if at this time it is inappropriate or painful, resulting in elimination irregularities or other proctologic symptoms.

To quote from Dr. Karl Menninger's book, Man Against Himself, "Where responses to emotional stimuli are irrepressible, the patient falls back to the more primitive spillway of the autonomic system—'they say it with symptoms.'"

The psychologic meaning might not be recognized at a future date when the symptoms have become habitual. Occurrence of a bowel movement caused by an acute emotional experience is well known and understood by all. The continuous reaction to chronic stresses is just as real but frequently unrecognized.

I would like to say a word here about the gastrocolic reflex. If the patient can revert to his childhood to his undoing, the physician may do likewise to his patient's advantage. The gastrocolic reflex has been suppressed by training and customs of civilized social life. In the constipated patient it may often be successfully revived and retrained with resulting regular evacuations after meals.

In all these problems we should not underestimate the help of the family, especially in younger patients, and give pertinent instructions to an intelligent member as far as feasible.

The extent to which psychoneurotic symptoms could mislead the profession is illustrated in a humorous manner in a discussion by Dr. P. W. Brown, at a Mayo Clinic staff meeting in 1932, about "the organic approach of a psychoneurotic bowel." Under the title "Doctoring the Bowels" he described uncountable symptoms and innumerable, inappropriate and unsuccessful treatments and operations of a hypothetic, typical gastrointestinal neurotic

which I think every proctologist and gastroenterologist should read.

We have all believed for a long time, more so after this war, that we cannot ignore the psychosomatic aspects of proctologic symptoms and diseases and will have to enter this field more and more if we are not going to deal with local pathologic conditions only but study the patient as a whole. More extensive knowledge of this subject will enable us not only to successfully take care of many of these patients but also to select more intelligently the patients with severe forms of the disorders who definitely require psychiatric attention.

ENDOCRINE dysfunction may cause substances to enter the circulation and thus increase sensibility of sensory nerves, leading to itching in the presence of local irritation. Thus diabetes, exophthalmic goiter, and the menopause must be considered.

EVALUATION OF ANORECTAL COMPLAINTS

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IT is frequently considered that the history is of secondary importance to the physical examination and without doubt this is often true, particularly if the history is hurriedly taken and improperly evaluated. However, in every patient's story there are pearls waiting to be plucked if the physician will take the time and effort to do so.

The first two sentences in Volume 1 of Gastro-Enterology by Bockus are: "The most important part of a clinical examination of patients with complaints related to the digestive tract is a good history. Obviously, the value of the history depends upon the intelligence of the patient and the keenness and experience of the historian." The medical student is taught to take a complete and detailed history although he has little experience in evaluating its salient features. In contrast, the busy physician frequently takes so much for granted and makes the history so brief that the patient's complaints serve as a basis for his diagnosis. It appears that following internship very little emphasis is placed upon the value of the history as there is a paucity of articles on this subject and very few text or reference books contain more than the advice to take a thorough history.

Before evaluating a history a good one must be obtained. The object of the anamnesis is not simply to fill in the answers to a list of prepared questions on a printed sheet, nor to question the patient regarding his concept or judgment of the case; nor is it to inquire what he thinks is the diagnosis. The only object is to supply the physician with a set of facts, from which he draws conclusions, based upon his knowledge and experience. A good history need not necessarily be long and detailed. The patient should have the opportunity to tell his story in his own words, for he then feels he has supplied the reason for his visit. This story must be used as a base line or the point from which to seek facts regarding the patient's problem; since especially in proctologic cases, the patient is often unable to state them clearly himself. The time spent in history taking allows the physician the great advantage of sizing up his patient as well as obtaining the latter's confidence. While I am not unmindful of the necessity of personally taking a complete history of every patient, or ascertaining that this is done elsewhere, this discussion is limited to the evaluation of anorectal complaints.

Obtaining the proctologic part of a history is somewhat different from that of the general history and must be evaluated accordingly. Several reasons for this are to be considered, the first and most important of which is terminology.

Proctologic terminology is so relatively new that many patients are not familiar with our terms and, if questioned, the answers will be vague and meaningless or entirely wrong, thereby misleading the unsuspecting physician in the diagnosis. It is obvious that if patient and physician speak different languages, no history whatever is obtainable and it should be equally clear that if the physician speaks in terms which are unknown to the patient, he will not be able to give an intelligent or correct answer. Many patients hesitate to admit they are ignorant of terms used by the physician and in preference to so stating will answer incorrectly. A short time ago I heard a resident in a hospital ask a patient, "Have you had any recent inflammatory proctologic condition?" Fortunately he received the reply, "Doctor, I don't know what you mean." But how

often would a question like that bring forth the easiest way out—a simple "NO"!

We think in proctologic terminology and are very apt to assume that the patient knows the meaning of our terms which is far from the truth. It is not too unusual for patients to deny having hemorrhoids, but to admit having "piles"; in fact, a great many patients' only conception of anorectal disease is "itching and bleeding piles." Also, consider the relatively old and familiar word "diarrhea"; how many patients know exactly what is meant by this term? It is too varied in its interpretation to be used directly to a patient, for what is "diarrhea" to one, has an altogether different meaning to another. Rather, receive an immediate and accurate reply to the query as to whether the patient ever has had four to twelve soft, watery stools daily. This correct answer may then be placed on the history sheet after the word "diarrhea" if the printed form is used. Therefore, since the patient is unable to interpret scientific terms of the proctologist, the latter must question the patient in his own language and in turn evaluate this in proctologic terminology. If the keenly alert and tactful physician suspects this to be the case, he will grasp the opportunity to assist with well chosen words, allowing the patient to select those which best suit his symptoms. This initiative will be productive, not only of a good and accurate history, but also of a grateful patient; otherwise he may leave the office with the impression that the physician is at fault for not diagnosing his case.

The patient is usually more reluctant to state anorectal complaints to the general practitioner than to the proctologist because, by the time he reaches the office of the latter, he is mentally prepared either by the first physician or by the severity of his symptoms "to go through the worst." Even so, many patients find it difficult to state frankly their complaints to the proctologist. The most usual reason for this is embarrassment since the patient regards his anorectal area as his private property

and its examination, either by the patient himself or by the physician, is uncommon. This feeling is enhanced by the fact that the rectum and anus act as a passage way for body excretions, and, in the presence of pathologic disease its cleanliness is difficult and uncertain. Patients frequently show this attitude by their remarks that they dislike to ask a physician to make what they consider an unpleasant examination, forgetting that the physician's interest is one of ascertaining facts regarding his patient.

Not only is the patient reluctant to make complaints of anorectal distress and submit to a proctologic examination because of modesty, but frequently fear of pain is a contributing factor. This may arise from his own personal experience with a painful anal ulcer, a cryptitis or external thrombotic hemorrhoids, or may be present because of the many and usual stories regarding painful anorectal examination, treatments or surgery. Whether or not justification for this view exists, it is a definite factor in many instances for withholding information regarding anorectal symptoms. To overcome this attitude requires tact, reassurance and patience on the physician's part and time spent in this way pays big dividends during the examination.

To be of value, a properly taken history must contain facts which are usually established only by careful questioning. If one accepts the history as told by the patient, much will be omitted because of the limited vocabulary, the intentional or unintentional oversight of some symptoms or the patient's poor recollection of forgotten ones. These omissions may very well contain the crux of the story and will be obtained only by careful and persistent questioning by the physician; in truth, this may be compared to an oral examination or even a cross-examination which implies a keenness of insight into possibilities and a persistency of inquiry into details to obtain all the facts present. To have a productive line of inquiry the

physician must be trained to think in terms of anatomy, physiology and pathology. In this manner his reasoning will quickly correlate the patient's complaints with known possibilities and his inquiries will be guided along logical lines and in the proper direction. Thus, there rapidly will be obtained a maximum of pertinent facts and a minimum of irrelevant material. Accepting at face value statements made by patients becomes a matter of accepting their judgment. The latter depends upon intelligence and experience and most patients have relatively little proctologic knowledge or experience and therefore cannot be expected to form correct opinions.

A few months ago a patient told me she had consulted a physician with the complaint that she had "bleeding and painful hemorrhoids." The busy physician immediately told the nurse to put the patient on the table and he would treat her hemorrhoids which he proceeded to do without further history or examination. The patient really had an anal ulcer but she made the only diagnosis she knew and, unfortunately, the physician accepted her faulty judgment as a fact.

In another instance, following the removal of a couple of skin tags, a patient called by phone at night, stated that she was having a severe hemorrhage and demanded admittance to a hospital. Upon arrival there only a small amount of bleeding was present although the patient persisted in the belief that she had had a severe hemorrhage. The color of the skin, pulse, blood count and blood pressure in conjunction with the local examination belied even any moderate hemorrhage. This then was only the patient's judgment.

These two illustrations could be multiplied by any of us and would serve only to center our attention on the truth of the statement that a patient's judgment of anorectal pathologic symptoms is unreliable. One is impressed by the frequency with which patients arrive at false conclusions because of lack of knowledge and

experience or because of preconceived notions. The physician must be on his guard against accepting these without due proof.

Referred symptoms are most difficult for the patient to recognize and interpret as well as for the physician to discover and evaluate. The patient usually is unaware of the possibilities that rectal or colonic disease may cause symptoms in other parts of the body and cannot be expected to appreciate the value of them in relation to their proctologic complaints. Consequently, they are often overlooked by the patient when he tells his story. But in searching for all the facts which can be elicited, the alert physician will be cognizant of these referred symptoms knowing that the rectum and colon are innervated by the sympathetic nervous system and consequently prone to present referred symptoms. The correct interpretation of these is essential and of inestimable value in directing the examination along productive lines.

In conclusion, it may be stressed that proctologic complaints can be evaluated properly only after obtaining all the facts present by the purposeful questioning of the patient; that this, together with physical examination and indicated laboratory work will lead to a correct diagnosis, proper treatment and a satisfied patient.

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DISCUSSION

KNOX BRITTAIN (Rochester, N. Y.): I agree with Dr. Brown's value of the history in proctologic cases. On the other hand, it does not displace or replace complete, adequate and thorough physical examination. Anyone who tries to make a diagnosis with either the history

PYRIBENZAMINE

ITS RÔLE IN THE TREATMENT OF PRURITUS ANI

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COMPARATIVELY few individuals suffering from diarrhea, prolapsing hemorrhoids, chronic discharging fistula, incontinent sphincter, etc., complain of pruritus ani despite the insult to the exposed integument by fecal material and malodorous infected discharge. The perianal region of over 1,000 young men examined for the draft showed contamination by varying amounts of excrement and yet very few complained of itching.

Is it not logical to assume that in pruritus ani there must be two contributing factors? (1) Sensitization of the skin; (2) an irritant (allergen or antigen). If this premise is true, then the pruritus must, in the main, be an allergic phenomenon.

"Recent investigations[1] have shown that all individuals are potentially capable of developing allergy. The capacity to become allergic depends on two fundamental factors: (1) The predisposing or auxillary condition[1] which paves the way for the allergy by making the organism prone to allergization. (2) The exciting allergens, in the case of food hypersensitiveness, the nutritional allergens which actually elicit the allergic reaction.

"Such predisposing factors are hereditary, the functions of the endocrine glands and of the autonomic nervous system, infection, intoxications, gastro-intestinal disturbances, hepatic disease, infestations, metereologic and climatic influence, social and environmental conditions, psychic influences, vitamin deficiencies."

A comprehensive history, intimate study of the involved integument, supplemented by skin tests if necessary, will determine to what group the exciting allergen belongs. The following questionnaire[2] is used in our practice:

I. Does the patient know of anyone in his family with: (a) asthma? (b) hay fever or vasomotor rhinitis (seasonal occurrences)? (c) atopic dermatitis including infantile eczema? (d) seborrheic conditions (baldness, acne, seborrheic dermatitis)? (e) hives—angioneurotic edema (giant hives)? (f) other skin eruptions, including those due to drugs? (g) other untoward effects from drugs? (h) other familial diseases?

II. Has the patient had: (a) asthma (not cardiac)? (b) hay fever? (c) stopic dermatitis (including "infantile eczema")? (d) fungous infections? or eczematous "ids"? (e) poison-ivy dermatitis? or other contact-type dermatitis? (f) seborrhea: 1. dandruff? 2. acne? 3. falling hair?—baldness? (g) hives? (h) other skin eruptions? (i) other diseases?

III. (a) When did the eruption begin? (b) Was there a previous similar attack? (c) Where did the eruption begin and how did it spread? (d) Is the eruption constant or periodic of sudden or gradual onset? 1. seasonal influences? 2. Other remarks on course of dermatosis. (e) Is there anything which has been recognized as making the skin condition worse? 1. foods? 2. clothing? bedding? articles of furniture? dusts? sprays? particular places of work, of residences, etc? 3. menses? 4. illnesses and remedies? 5. miscellaneous? (f) What treatment has been used—internal and external medicaments? (g) Medication in general (proprietary or prescribed): 1. for headaches? 2. for constipation? 3. for "acid in systems" (blood purifiers," tonics)? 4. for sleeplessness? 5. for "nervousness"? 6. for coughs? 7. for dysmenorrhea? 8. for "sinus trouble"? 9. Bromo Seltzer? Bromo Quinine Laxative? Ex-Lax? phenolphthalein in any other forms? other "patent"

remedies? home remedies? medication on recommendations of relatives or friends, of druggists, of previous physicians? (10) iodized salt? (11) other medication—internal or external, proprietary or prescribed? (h) Cosmetics 1. used by the patient? 2. used by others in the patient's environment? 3. applied at beauty parlors or elsewhere? (i) Occupational and avocational contacts: 1. At work (a) principal substances handled? (b) incidental substances handled (cleansers, washing materials, "protective" materials, etc.)? (c) physical condition of the contacts? (heat, moisture, friction) 2. At home (a) cleansers, insecticides (professional exterminators), (b) pets, plants and substances used in their care, (c) clothing, furniture, drapes, (new-cleaned—dyed)? (d) hobbies—photography, stamps, carpentry, gardening, golf, cards, other games? (e) miscellaneous? newsprint, rotogravure, jewlry, nickel-plated articles, rubber articles, etc., etc.,

Summarizing this history form, the exciting allergen will be found to fall into one of the following groups:

1. Allergy to food: Shell fish, fish (including caviar, etc.), strawberries, cheese, nuts, eggs, wheat, milk, pork, chocolate, cocacola and alcohol in all its forms, etc.

2. Allergy to fungi and bacteria: Trichophyton, epidermophyton, or monilia albicans, any bacteria.

3. Contact dermatitis: (Lesion primarily epidermal; spongiosis, vesicle, papule, etc.) dyes, silk, wool, perfume, toilet water, talcum powder, cleaning fluid, drugs and ointments applied to the skin, leakage of mineral oil with fecal material, feces, clorox used in bleaching clothes. Sensitivity to fecal material is probably due to skatol, indol and complex phenols. The medicinal agents applied in extended postoperative care will often cause a contact dermatitis. Benzocaine is a frequent offender.

4. Allergy to drugs: Any drug taken internally may cause symptoms. Such drugs are ipecac, quinine, barbiturates, opiates, iodides, including iodized salt, aspirin, bromides, antipyrine, sulfa compounds, phenolphthalein, etc.

5. Allergy to atopans—atopic dermatitis: Specific circulating antibodies can be demonstrated by passive transfer experiment of Prausnitz and Kuestner. A complex of symptoms and phenomena which can be explained primarily by increased vascular permeability. A family history which is positive for asthma, hay fever, atopic dermatitis and perhaps certain other diseases such as migraine, hives and gastrointestinal manifestations.

A personal history which often reveals, in addition to the presenting atopic disease, certain other atopic manifestations—mentioned above. An eosinophilia in the blood smears and/or in the fluids and tissues of the affected parts. A specific substance (called atopen) which reaches the sensitized cells in sufficient quantity. (The atopens may be inhalants, such as: pollens, spores of fungi, emanations from animals, insects, etc., dusts and powders, orris root, rice; ipecac, caroid and lycopodium in prescriptions filled by druggists, wheat flour, etc., either by inhalation, swallowing or by external exposure and transepidermal penetration of the outer skin.) Cosmetics, insecticides, etc. Injected substances—serums-vaccines, etc. Microorganisms including bacteria and fungi. Drugs (aspirin, etc.). Foreign proteins. (All these can act by any route provided they reach the specifically sensitized tissue in sufficient quantity per unit of time.)

The allergic individual has sensitizing antibodies circulating in his blood. These are also attached to the body cells of a certain organ known as the shock organ. A foreign substance (irritant, allergen, antigen) is introduced to the skin or in the body which interacts or probably combines with the antibody liberating histamine proper or a histamine-like substance caus-

ing the allergic reaction by breaking off a CO or carboxyl radical from histidine, one of the body amino acids.

There is hardly a tissue that will not respond in some way to histamine.[3] This drug is a powerful dilator of capillaries. As a result there is a loss of plasma protein and fluid through the capillary wall into the extracellular spaces. This phenomenon is most important in pruritus ani. All histopathologic studies of pruritic skin emphasize marked dilatation of the blood vessels.

"PYRIBENZAMINE (N'-Pyridyl-N'-Benzyl-N-Diethyl-Ethylenediamine HCL) was discovered by Mayer[4] and his associates. It is a white crystalline material which is stable, non-hygroscopic and readily soluble in water. It can be safely administered in dosages up to 150 mg. four times a day preferably after each meal and at 10 P.M.

"PYRIBENZAMINE is well absorbed[5] from the gastro-intestinal tract, peritoneal cavity, subcutaneous and intramuscular depots. Its oral administration is so satisfactory, that the risk of unwanted side reactions from parenteral administration is unwarrantable. Nothing is as yet known of its destruction, deposition or secretion."

Its use over a considerable period of time has as yet not evidenced any effect on the blood, urine, liver or other vital organs.

As to the action of pyribenzamine, I should like to quote from a letter received from the Research Department of the Ciba Pharmaceutical Products:

"Frankly, we do not know exactly how pyribenzamine acts as an anti-histaminic agent, but it is safe to state that pyribenzamine and histamine enter into a competitive relationship in that they compete for the site of action normally involving histamine whether it be in a muscle, glandular or other type of cell. We speak of this phenomenon as that of competitive inhibition and you are correct in your statement when you say that pyribenzamine acts better when employed prophylactically than correctively; in other words, an ounce of prevention is worth a pound of cure seemingly.

"Pellerat[6] has demonstrated that after the administration of antihistaminic agents to experimental animals not only is anaphylaxis prevented but histamine is liberated in abundance. This indicates that histamine does not seem to be chemically neutralized or physico-chemically fixed with the anti-histaminic agent but rather that it is prevented from eliciting its normal physiologic reactions on the part of the system due to the fact that it apparently is insulated against by pyribenzamine or another antihistaminic agent at the site of its normal action whether it be at or within the cells involved. This insulation, probably similar to that afforded the electrician against electrical currents by the use of the rubber glove, might be purely mechanical in the sense that a protective physical relationship is established at the surface of the cell, but in the light of modern biophysics and biochemistry, one would anticipate that certain enzymes might be involved in this relationship. What these enzymes are in problematical and only time will permit elucidation of this phenomenon."

Dr. Rudolph Baer, of New York, gave a quantity of this drug to my wife in 50 mg. tablet form to alleviate a distressing, itching, allergic foot condition. The tormenting itching and swelling was promptly relieved.

If this new agent were to control the itching of the majority of patients with pruritus ani, the topography of the skin would improve because the trauma of scratching would be removed. Sleepless nights would end, the patient's health and morale be bolstered and skin therapy would provoke a greater response. It would above all demonstrate that the pruritic skin lesion in the perianal region and its environs is the result of an histaminic action otherwise why could it be controlled by an anti-histaminic drug? Since most allergists agree that reaction in the receptor cells of the shock organ has to do with a histamine effect, such a response to specific therapy would prove our premise that pruritus ani is in the main an allergic manifestation.

This research with pyribenzamine began in July, 1945. The supply of the drug was limited at that time so that our experiment began in earnest in November, 1945.

A careful history was taken from each patient using Sulzberger's questionnaire. The patient was instructed to write down each day foods consumed, drugs taken and note particularly if anything partaken internally or used externally influenced the itching. If the incriminating allergen could be determined, it was eliminated.

Patients with pruritus ani were advised to take 50 to 150 mg. of pyribenzamine after each meal and at 10 P.M. They were warned against any other medication for at least forty-eight hours. Placebo tablets provoked no relief when prescribed. They were not told what the action of this new drug would be so that the psychic effect could be discounted.

If drowsiness occurred, the dosage was reduced. Drinking black coffee would usually overcome this symptom. If any untoward syndrome developed, the drug was discontinued.

Dosage could be reduced to one tablet a day. If the individual complained of nocturnal pruritus, an appropriate quantity was given after the evening meal and before retiring. Gradually, one tablet before retiring was frequently sufficient to insure an uninterrupted night's sleep.

Because Gerhard Katz[7] and other observers were able to demonstrate histamine in allergic skin lesions an attempt was made to evaluate the effect of a 2 per cent pyribenzamine ointment* in a water washable base, applied to the involved area.

When first applied, the ointment causes a smarting sensation lasting about one minute. If it is effective, the itching should be controlled within ten minutes. In this survey it was revealed that only those patients that responded to pyribenzamine taken orally obtained relief when the ointment was applied to the skin. After a time the itching could be controlled by local therapy alone.

* Supplied by The Ciba Company.

Our best results were obtained using a combination of oral and local medication. Adequate doses given by mouth yielded better results than when only the unguent was used. Patients with an acute, exudative eczema of the perianal region were not treated with this ointment.

If the incriminating allergen had not been ascertained, further studies were made but in the interim the patient was given the following instructions:

(1) Omit from the diet anything sour, bitter, salty, condiments, flavors, coffee and all forms of alcohol. Mineral oil or oil base medications must be avoided.

(2) After defecation wipe perianal region with wet absorbent cotton, sprinkle acidolate on cotton and wash the affected area (to remove sweat, fecal material and ointments). Wash off acidolate with another portion of wet cotton and then dry with absorbent cotton. The area is then covered with either lotion, unguent or powder, depending upon the skin topography. Always keep the area covered with a thin layer of fine, absorbent cotton. Some may argue that this cleansing is responsible for the relief and cure of the pruritus. In my series this was true in only six patients.

(3) Do not take tub baths, only showers.

(4) If interdigital epidermophytosis is present, soak feet in hot water for ten minutes to remove sweat and dye from stockings. Dry feet thoroughly. Apply liquid sopronol on an applicator between the toes and to other affected portions of the feet. This should be done at night. In the morning, dust powdered sopronol between the toes and over the affected areas. Wear white cotton stockings. A separate towel should be used to dry the feet so as to avoid transmitting infection to the perianal region or groin.

(5) In patients with acute exudative eczema or when the itching is tormenting in character, ice in a throat ice-bag applied to the perianal region will usually give relief in fifteen minutes. In those cases in which there is weeping, a 1 to 20 liq. burowii compress is applied to the skin and the ice-bag

placed over it. This freezing agent must be pressed closely to the compress to avoid a column of air between ice and skin. A domeboro tablet dissolved in a pint of water will make a fresh 1:20 solution. Application of ice numbs the nerve ends and constricts the capillaries and arterioles. When the acute skin phase becomes subacute or chronic, appropriate lotions, unguents or powders are applied.

(6) In patients with contact dermatitis from feces, cover the perianal skin with zinc oxide ointment before evacuation. After the bowel is emptied and the perianal region washed with acidolate cover the integument with 1 per cent methol in equal parts of zinc oxide ointment and desitin ointment.

(7) If skin is infected or undermined, supply adequate drainage.

(8.) All clothing or bed linen coming in contact with the perianal skin should de washed in lux. Clorox or other bleaching agents used by commercial launderies frequently cause contact dermatitis.

Young women suffering from profuse vaginal discharge were routinely examined for trichomonas vaginalis. If a history of pinworms is elicited, appropriate therapy should be instituted. Urine is examined for sugar. Metabolic diseases, vitamin deficiency, etc., are adequately treated.

STATISTICAL DATA

Ninety patients were treated with pyribenzamine. Fifty-seven had satisfactory results (85 to 100 per cent had relief from itching); twenty-six cases were due to food allergy; ten cases were due to fungous allergy; twelve cases were due to contact dermatitis; three cases were due to atopic dermatitis and six cases were due to drug allergy. Eleven patients reacting favorably to the oral administration of the drug were controlled by the local application of a 2 per cent pyribenzamine ointment in a water washable base. This ointment was only available for the last few months of this survey.

Thirty-three patients had unsatisfactory results. Sixteen did not return after their first visit, the investigation was incomplete and the response to therapy was uncertain; there were four patients in whom the drug had to be discontinued; two patients developed a generalized maculopapular eruption; one patient complained of cardiac palpitation and extreme nervousness; one patient developed very profound drowsiness; six patients with contact dermatitis were not controlled; in the four patients with senile pruritus three responded to estrogenic and androgenic therapy; one patient was diabetic and two patients had drug allergy.

There were two patients with psoriasis principally involving the posterior aspect of the perianal region and extending toward the coccygeal area. The only other phase of this disease was found under the folds of the mammae. These patients were relieved by using a $\frac{1}{2}$ to 5 per cent ammoniated mercury ointment and finally healed with a 3 per cent vioform unguent in a water washable base.

Arbesman[8] and his associates were the first to conduct therapeutic trials with pyribenzamine. Feinberg[9] reports excellent results in controlling the itching of pruritis ani and vulvae.

CONCLUSIONS

1. Anorectal pathologic conditions play an insignificant rôle in the etiology of pruritus ani.

2. Pyribenzamine will not cure this syndrome.

3. Pyribenzamine will control itching in the majority of patients. By reducing sleeplessness the morale and general health of the afflicted person improves. Since the trauma of scratching is removed, the pathologic condition of the skin improves and adequate therapy can be instituted (lotions, ointments, powders, etc.).

4. The percentage of patients with pruritus ani due to fungous infection is not as high as anticipated. Because there is an interdigital epidermophytosis in the same individual, it does not prove that the skin

lesion in the perianal region is necessarily of the same origin. It is the exception rather than the rule for the patient with athlete's foot to suffer from pruritus ani.

5. Careful investigation will clarify the etiology of many patients heretofore classified as idiopathic.

6. If the incriminating allergen has not been ascertained, it is due either to the fact that the investigation was incomplete or because our methods of determination are thus far inadequate.

7. Because a considerable percentage of patients do not respond to pyribenzamine it does not rule out their allergic origin. The logical explanation must be that the pyribenzamine is unable to insulate the receptor cells in the shock organ against the action of histamine. This fact is further borne out by the more satisfactory results obtained in animal experiments when pyribenzamine is administered prior to exposure to the offending allergen.

8. The degree of success in the alleviation and cure of pruritus ani will depend on the thoroughness of the method of investigation plus a fundamental knowledge of allergy, dermatology and proctology.

9. Pyribenzamine merits a definite and important rôle in the treatment of pruritus ani.

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RATIONAL OF THERAPY IN PRURITUS ANI

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WE are all hoping that some one of us will rise and deal pruritus ani one fell blow that will knock it out of the ring but to our sorrow the time is not yet at hand. There is such a maize of interlacing and overlapping of factors that even cause and effect become reversible.

Pruritus ani as it confronts the proctologist is not a mere symptom but constitutes a definite syndrome[1-4] and along with the recurrent attacks of itching in the ano-perineal region, we are involved with the associated symptoms of anospasm, insomnia, nervous depression and sluggish peripheral circulation; also excessive perspiration in intertrigenosis areas, a moderate leukopenia and a subclinical nutritional deficiency state and, of course, the gamut of skin involvement.

Whether or not we accept pruritus ani as a syndrome we are all, nevertheless, aware of the coexistence and importance of the associated findings. Our rational of therapy, therefore, must include the consideration of all these factors.

The nervous manifestations are so characteristic that we can easily describe a type that would represent a cross section of these patients. They look, as a rule, well nourished but pale and pasty with a grayish cast to their skin. Even when thin, they are sluggish and slow moving with lackluster eyes. They are generally not communicative but give an impression of dejection at times amounting to a state of despondency with suicidal tendencies. These nervous manifestations represent a vagosympathetic imbalance of the vagotonic type.

In order to appreciate the psychosomatic factors of this syndrome we must review the nervous mechanism involved. It is true that the itching sensation travels by way of the sensory or pain fibers, however, the threshold of irritability for receiving the sensation involves the vegetative (autonomic) nervous system so that when the vagosympathetic equilibrium is upset, as in vagotonia, a severe pruritus may be elicited by ordinary innocuous stimuli. Furthermore, since the psychic factor is the true receptor of the sensation, its state of equilibrium will in turn determine the degree of response it will give to the sensation of itching.[5]

Any therapeutic measure, therefore, that tends to calm the psychic state or reduce the nervous irritability[6] or tissue sensitivity enters as a valuable factor is the treatment of pruritus, whether it is bromides, barbiturates, phenobarbital or calcium or the more recent anti-hystamine preparations like benadryl and pyribenzamine.

It is further established that some of these states showing autonomic imbalance have their basis in vitamin B deficiency.[7,8] I have long recognized the importance of these factors, so much so that when I find the pruritus associated with a vitamin B deficiency I consider the problem half solved.

The basic pathologic condition consists of three factors: skin irritation, anospasm and lymph stagnation or lymph edema. These three factors are so completely interlaced that you cannot separate any one from the others. Together they constitute both the cause and effect of neurobegetative imbalance and form the groundwork of this syndrome.

RÔLE OF LYMPH STASIS

Lymph stasis brought on by the slowing of the peripheral circulation plays an important rôle in the chronicity of pruritus. It allows the further accumulation of the

irritating substance whether it is excessive or abnormal products of metabolism or actual toxins.

Hanes[9] began his local injection procedures when he recognized that there was some disturbance in the cellular metabolism in the local tissue. He described the almost immediate correction of advanced skin changes following his injections in the subcutaneous tissues. Those of us who have used the Hanes procedure have witnessed the disappearance of even remote coexisting skin lesions following the injection of hydrochloric acid solution in the perianal region.

Our injection methods, even those that primarily anesthetize the sensory nerve endings, all produce a phagocytic response and basically have the same physiologic effect of decongesting the peripheral circulation. The phagocytic activity of the histeocytes produced by the reaction to these injections are known to remove toxins and protein particles as well as bacteria so that they may account for the major part of the readjustment of peripheral lymph circulation.

Another very probable factor is the interference with adequate oxygenation of the tissue cells by the slowing of the circulation. This may be the reason for the good results obtained by direct injection of oxygen as described by Dr. Guess.[10]

We see, therefore, that there is a common denominator in the rational of all our injection treatments.

RECTAL PATHOLOGY

We all agree that the more common rectal conditions are seldom directly responsible for the itching. However, the most constant rectal finding in all these patients is anospasm. Any indication of a rectal pathologic condition that will produce or intensify rectal spasm should be corrected for if it is allowed to remain this condition alone can eventually bring about neurovegetative exhaustion.[11]

It is always best to postpone surgery until we can determine the underlying factors that are interfering with the normal physiologic balance. Proper timing of drastic procedures is also of value for the morale of the patient. It is important in case of recurrence to prove to the patient that the itching can be relieved by simple measures and that our clinical resources are not easily exhausted. However, my only failures are those when I underestimated the despair of the patient and waited too long before instituting drastic measures for his relief.

In frustrated individuals who are facing insurmountable problems and in patients who have received x-ray therapy, it is best to give immediate symptomatic relief and proceed later with general measures to correct basic underlying factors and with instituting measures to prevent recurrence.

However, I see no justification for radical surgical procedures *per se* and injections involving mass tissue destruction. I do not even use the undercutting type of operations because I find that alcohol injections by the multiple puncture method of Stone[12] and Wilson[13] will accomplish just as much. It is rarely necessary to repeat the injection more than twice.

Whenever there is scar tissue, redundant folds, pectinosis, marginal varicosities or ulcer scars with thickened margins, I find it advisable to perform surgery and use alcohol stippling to prevent recurrence of pruritus immediately following operation.

RÔLE OF FUNGUS INVASION

One of the features of this syndrome is the excessive perspiration of the apocrine sweat glands, producing an alkaline secretion rich in protein and carbohydrates. The location of these apocrine glands are also and probably therefore the site of predilection for cultivation of fungi. Since they are saprophites, they are also furthered by the basic interference of the cellular metabolism by the lymph edema described above producing partially devitalized cells more suitable for their saprophitic comsunption.

Once they arrive the fungi entrench

themselves by means of their alkaline secretion and perpetuate and intensify the very same cultural conditions that made their arrival possible.

It is very difficult to isolate the fungus organism from the perianal skin once treatment has been instituted.[14] An important factor to take into consideration is the seasonal tendency of recurrence: Not only is there a much greater incidence of cases during definite periods but the organism can be detected much more readily during that time. The beginning signs of fungus pruritus is the linear abrasion or scaling in the intergluteal fold. I call this the "tell-tale line." Another tell-tale sign is the thickening or lymph edema of the anterior median raphé in the male.

In advanced cases as we all know the skin undergoes a great variety of changes. We must remember that conflicting medications can give rise to an "hysterical skin" so that on first inspection it will not fit into any category. At this stage it is best to use the so-called bland and soothing medications such as boric acid ointment. At times the irritation is so extreme that the skin will exhibit a non-specific sensitivity reaction to almost all topical medications so that only bland oils will be tolerated. This is a critical point in the progress of the patient with pruritis. I call this the "crisis." It is at this stage that the patient becomes frantic and changes doctors or the doctor becomes frantic and resorts to irrational procedures.

We must remember that all fungicidal ointments owe their effectiveness to their acid nature. However, fungicidal ointments that have a heavy petrolatum base will only plug the hair follicles and sweat glands of the perianal region and will not reach the fungi. Such an ointment, although most effective in other parts, will only cause further irritation of the perianal skin. In these instances it is best to use penetrating dyes.

In conclusion I want to emphasize that we must accept the rôle of the clinician in addition to treating the rectal pathologic condition if we want to track down and irradicate pruritus ani tendencies. I credit my good results to the fact that I accept pruritus ani on its own terms and give all features of the syndrome equal respect.

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DISCUSSION OF PAPERS OF DR. FRANK M. FRANKFELDT AND DR. RACHELLE SELETZ

ROBERT V. TERRELL (Richmond, Va.): While in general agreement with the views expressed by Drs. Frankfeldt and Seletz I wish to re-emphasize my own belief that the cause of pruritus ani is in most cases a mycotic infection of the perianal skin. I readily concede that allergy plays a part in many patients just as a neurosis undoubtedly is a factor in a few.

Best results will be obtained by modifying the treatment to suit the condition of the perianal skin and the character of the patient. Should an allergy or a neurosis be present, efforts are made to correct these while treating the skin. After a painstaking proctologic examination considerable time is spent in explaining to the patient in language he can understand just what is wrong with him and how we shall attempt to correct the condition. The importance of his cooperation is stressed, explaining that he should use only drugs prescribed to prevent the possibility of a drug dermatitis. He is also told that treatment must not be discontinued as soon as the itching ceases but kept up until the skin returns to normal.

The patient understands well enough that scratching aggravates his condition and if it is thought that local applications will not adequately curb the desire to scratch, a few cc. of 0.25 per cent diothane hydrochloride are injected beneath the involved skin. Relief from itching after this procedure is usually prompt and lasts several days. Every effort is made to improve the condition of the skin while this relief lasts.

Many patients when first seen have an acute inflammation or show a drug dermatitis. It is important in this type to eliminate any irritant and use soothing measures such as boric acid ointment or even hot boric acid compresses. Pyribenzamine is particularly useful in cases of this kind. Careful cleanliness is important in all cases but soap and water are best avoided in exudative conditions.

In the more chronic types, 2.5 per cent undecylenic acid or a diluted Whitfield's ointment may be applied three times daily. A few fibers of dry cotton interposed between inflamed skin surfaces will reduce friction and irritation and absorb serious discharges.

When pruritus ani is complicated by fissure, fistula or hemorrhoids these conditions are corrected by surgery. The patient is made to understand that we are not operating primarily for the pruritus even though we do expect it to improve. Treatment for pruritus is continued after the operative wounds have healed. We have never thought it necessary to undercut the perianal skin.

HARRY C. GUESS (Buffalo, N. Y.): The person discussing a paper, or in this case papers, has two alternatives. He can agree and comment or disagree and augment.

Pruritus ani, our perennial visitor, is a large and one of the most interesting subjects in proctology. For many years each year something new has been added to our already large armamentarium for the treatment of pruritis ani and often each one who offers, and some few followers, are lulled into a complacent state that the last word for pruritis ani has been spoken. Like religion striving for the heavens, some by water, some by air and some by other means, we too continue to strive for the best "in the end."

Dr. Seletz voices the hope that quote, "someone of us will rise and deal the final blow to pruritus ani," unquote. We concur in that hope. There is no doubt that we agree on the associated clinical findings in most cases and our therapy must include the consideration of all factors. That there is lymph stasis as well as arterial and venous congestion is factual.

Her timely suggestion to limit radical surgery is well given and accepted. There is probably no one condition that has so many remedies offered as does pruritis ani. Her description, as the result of the use of many of these remedies, of a "hysterical skin" is well presented but to me some are not only hysterical, they are crazy. Her conclusion that the clinician "should track down and eradicate pruritus and its tendencies" is also excellent.

As for Dr. Frankfeldt's paper my knowledge of allergy is limited and I make no claim to understand the various phases of that condition. It is said that it is better to remain silent and be classified as dumb than to speak and remove all doubts. His is a contribution for the use of one antihistaminic drug with which he has good results with the help from chemo and low temperature therapy. He states that there are two contributing factors, namely, (1) sensitization of the skin and (2) an allergen as the principle cause of pruritus. He states that if this premise is true then pruritus must be, in the main, an allergic phenomenon. That is the sixty-four dollar question as yet unanswered. I cannot agree with this statement except in some cases. My concept regarding the pathologic conditions causing pruritus ani and our results from various medical and surgical treatments reported previously over a period of years is, I believe in the main, the cause of pruritus ani.

That end oxins, allergens, histamine and the end products of the metabolism, whether it be Carbon No. 1 or Carbon No. 13, do cause irritation of the tactal corpuscles in the nerve endings is true and an anoxic state results. These end products of metabolism no doubt remain in situ because of the slow lymph arterial and venous drainage.

Regarding the statistics which Dr. Frankfeldt quotes, I have had some experience with the antihistamine product in question. Much work was done by men from my home town on hay fever, asthma and urticaria but very little with pruritus ani, and the results which stated that twenty-seven or twenty-eight patients with pruritus ani were markedly improved by this antihistamine drug were reported by a specialist from some other city. Request for

information regarding these patients was not forthcoming because the contributor had not released his paper to the press. In the latest reports of this drug, pruritus ani is not reported. However, Dr. Frankfeldt in making his conclusions does not claim the utmost for the drug but states (1) "That pyribenzamine will not cure pruritus ani;" (2) "will control itching in the majority of cases" and (3) "that it has a definite and important rôle in the treatment of pruritus ani." With these statements we must agree except for the adjectives describing the rôle and the treatment.

To conclude, both contributors have given food for thought and are to be commended for their investigations. We should keep in mind at all times (1) That we must not accept each new drug without sufficient evidence that there is value to the drug mentioned; (2) that we, as proctologists, should have an open mind in the diagnosis and treatment of this condition and (3) that some day we, as an organization, will correlate with the physiologists, pathologists, psychiatrists and biochemists and bring to a realization the ultimate for which we are all striving—that "final blow to pruritus ani."

HOWARD K. BELNAP (Ogden, Utah): In discussing the papers of Dr. Seletz and Dr. Frankfeldt, Dr. Terrell and Dr. Guess have covered the subject as I would cover it very accurately. I am of the opinion that had Dr. Frankfeldt had a little more time in presenting his paper you would have heard a regimen of therapy that was very meticulous; it was bland; it was relatively non-irritating to the skin but I am sure that with or without the use of pyribenzamine, he would have obtained as good results as he is doing with pyribenzamine.

I want to reiterate that he does not attribute pyribenzamine as a cure, only as a relief of the itching; yet any drug that will relieve 77 per cent of patients with itching within twenty-four hours is worthy of trial. Pyribenzamine was first introduced to me in January. Like all of the other remedies that we find are at present on the market and as articles appear in the literature, we grasp at them as though we were a drowning man grabbing at a straw in a stream for we have as yet not found a specific therapy for the disease, pruritus ani. Since January, I have used pyribenzamine as directed in the literature and as outlined by Dr. Frankfeldt. My results have not been as striking as Dr. Frankfeldt's. I have obtained relief of itch-

ing in about 13 per cent of my patients. Maybe I did not use as much of the drug as was necessary to disrupt those inhibitor factors that Dr. Frankfeldt talks about but I used the prescribed milligrams four times a day.

Again, let me repeat I think his good results have been from his other therapy.

In Dr. Seletz's paper, the discussion that it is a vagotonic, vagosympathetic balance is really something to consider. She has brought forth a point that we have, as proctologists, been tending toward in the last two or three years. As you look over the review of papers presented, we are connecting up the sympathetic and the parasympathetic systems with the symptoms and conditions we find about the rectum. Her discussion is a step in that same direction.

She, again, outlines a regimen of therapy that is good. It follows along the same outlines as we generally apply. Whether she looks at all the other symptoms and treats each one of them with an individual pill or with some individual type of psychotherapy, that is a problem and her method of therapy and it should be good.

I agree with Dr. Terrell and am in accord with him to the extent that I believe pruritus ani has its basis in a micotic organism. In my patients I found either a trichophyton or one of the epidermal phytons in scrapings of the perianal skin. I think that sometime in the future when we are able to obtain a fungicide that will penetrate through the entire corneal layers of the skin without producing a deleterious effect, we will definitely come to some conclusion in the treatment of pruritus ani.

MALCOLM R. HILL (Los Angeles, Calif.): My short discussion will be brought up in the light of a few remarks alluding to Dr. Terrell's premise that mitotic origin of pruritus ani still stands in the foreground. My groundwork on pruritus ani was obtained from Dr. Emmett Terrell years ago. It was on his remarks to me as an individual and placing that premise in the foreground that a study and report was made to this organization at the Richmond, Va. meeting several years ago.

Upon visiting the skin practitioner or dermatologist, I asked him what the relationship between pruritus ani and the id manifestation of epidermophytosis was. I got a negative answer. Most men evaded me. In this report at Richmond, Va., we brought out the fact that 6 per cent of pruritus ani was of mitotic origin. We also broke that 6 per cent group into a 100

per cent base and alluded to the fact that only a fair percentage had a primary invasion of the perianal skin. That there was an id manifestation in a fair percentage of these individuals, an allergic manifestation, was also brought to light.

May I reiterate that id manifestation in the light of the dermatologist is a secondary eruptic or non-eruptive manifestation away from the primary focus of infection. That same type of phenomenon can be present about the perianal tissues from a primary on the feet, between the toes or the planar surface.

An allusion has been made to the fact that this treatment must be considered about the perianal tissues. No mention has been made in this general discussion of the importance of taking patients' shoes and stockings off and examining their feet along with the perianal examination. That is important.

Dr. Robert Terrell has alluded to the fact of the importance of perianal hygiene. May I reiterate that if you do not teach your patients how to cleanse the perianal tissues and keep them adequately clean at all times, literally being told they must bathe themselves after each evacuation for the remainder of their days, you are not arriving at one of the primary problems of treatment of prutitus ani.

Finally, and not least, the importance of eradicating distant foci of infection or primary focus of infection in other parts of the body, whether it be of an epidermophyton phenomenon or other allied skin problems, should be borne in mind.

FRANK M. FRANKFELDT (closing): As regards Dr. Guess' discussion, the results you are going to obtain in the interpretation and treatment of pruritus ani will depend on the thoroughness of your investigation, your knowledge of allergy and appropriate therapy if you have not found the incriminating allergin. There is no question about it and there is no fact to dispute the premise that if you find a pruritic skin there must be two factors: There must be a sensitization of the skin on the one hand and some irritant on the other hand. Otherwise, how can you explain the fact that every one of us sees thousands of patients with anorectal pathologic conditions when the skin is bathed with these irritating discharges and they do not itch. Well, why is it that other people do itch? There must be a rationalization for it. As ye seek, so shall ye find.

As busy proctologists, it is unfortunately much easier to take a Flit gun and shoot something under the skin than to spend a few hours investigating a case and treating the cause rather than the effect.

As regards fungus infection, I think no one has preached fungus infection more than I have. I think the members of the New York Proctologic Society will remember that quite a number of years ago I tried to emphasize the frequency of fungus infection as an etiologic factor in pruritus ani. In 1938, I had the privilege of collaborating with Sir Albert Castellani and we discussed the subject in detail. You do not frequently recover the epidermophyton either from the original focus of infection between the toes or from the id. Because a patient has athlete's foot and has pruritus ani, it does not necessarily follow that that patient has an id phenomenon; if you were to take the shoes and stockings off most of us here, we would be very much surprised how many people do have desquamation and evidences of infection between their toes. There must be a criterion. If a man like Castellani had examined one patient nineteen times before he could isolate the trichophyton or epidermophyton, how futile would be our attempts to try to get these organisms? Why have I included some of these patients under the caption "fungus allergy"?

Incidentally, when you get pruritus ani as a result of a fungus infection, you have a fungus allergy and you have a contact dermatitis which is caused by the products of metabolism of these fungi in the diseased skin, just the same as the other phenomena are allergy but, unfortunately, not understood. Now, to say a case is due to fungus, of course, the ideal thing is to be able to say, "I have found the fungus," which very few of us can say.

I know the work the elder Dr. Terrell did because I was in correspondence with him a number of years ago and at that time I was very much interested in fungus infection. Unless you have an acute exacerbation of an interdigital infection and then a perianal lesion, it is hard to say that that particular thing is due to a fungus infection.

It is hard to say that a perianal lesion is due to a fungus infection. If you had a tinea cruris and a pruritus ani, you would be more safe and more apt to be just in saying that that is a fungus infection because it was carried by a towel.

Gentlemen, there is one other point if I may make it. If you ever have an intractible patient, one who comes to you with agonizing itching, there is one thing that will stop the itching and you can bet your bottom dollar on it; that is ice. If you will take a throat ice bag and put it on a suspensory belt under the perineum, you will find that because of the contraction of the capillaries plus the freezing of the nerve ends, you will in almost every case be able to control the itching. If I had time, I could go into many more points.

RACHELLE SELETZ (closing): I want to thank the discussors for their very gracious comments. Everyone was right and everyone was wrong, of course.

In regard to the so-called id reaction of Dr. Hill, we all agree about the fungus; I mean most of us do. All of us seem to agree about incidence of fungus. It is right that we should look at the feet and look behind the ears or in the external auditory meatus or, if you please, even in the umbilical orifice because that is the usual sites for hiding away of fungi. Incidentally, it is not because we do not wash there but because we do not dry there.

When a person has interdigital epidermophytosis, it does not mean he particularly has an id reaction but it may mean that he is a vulnerable subject for fungi.

During the war many medical officers spoke about the incidence of fungi. A particular major with the combat forces described thousands of patients that he supervised, with only one instance of pruritus ani and that only in a soldier who did not want to be an artilleryman.

When you have the proper circulation, you do not get pruritus ani. This brings us back to our syndrome. One thing alone is not enough. The body mechanisms are not that simple. It is only when the proper neurovegetative imbalance is produced by preferably anal spasm, psychiatric factors with the hyperactivity of the perianal sweat glands, (as we discussed before), plus an overconsumption of carbohydrates, that the fungi have a chance to take hold of the skin and we will have pruritus as a result. It takes more than one contributing factor. You must consider pruritus ani as a syndrome to include all these separate factors.

MODERN SURGICAL TREATMENT OF HEMORRHOIDS AND A NEW RECTOPLASTY

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THE objectives to be sought in performing an operation for hemorrhoids are: first, to remove all pathologic tissue second, to preserve all the normal tissue third, to maintain or restore the normal anatomic configuration and thus to maintain or restore normal function fourth, prevent sequelae and complications and fifth, keep pain and morbidity minimal.

It is well to recall the special anatomy and physiology of these parts, the characteristics of the tissues and their type of healing when injured. The anal canal is an elastic, distensible and contractile tube surrounded in virtually its entire length by voluntary muscle with a sphincteric action, and in its upper two-thirds its wall also contains the involuntary sphincter. The lower portion of the lining is of ectodermal origin and in reality is modified non-secreting skin; the upper portion and the rectum proper is lined by mucous membrane which is of entodermal origin and secretes mucus. The line of junction of mucous membrane and modified skin is at the semilunar valves; it is 2 to 3 cm. from the anal margin. While connective tissue and epithelium regenerate, injured muscle does not regenerate but is replaced by relatively inelastic, non-contractile, fibrous tissue.

Stenosis, seepage and early recurrence of hemorrhoids are among the sequelae following hemorrhoidectomy. Their pathogenesis, briefly stated, herein follows: The stenosis may represent an actual narrowing or a loss in elasticity or distensibility of the tissues. The factors responsible may be the removal of too much circumference, damage to muscle and replacement by fibrous tissue and increased fibrosis as a result of infection. Seepage may be due to a downward displacement of the secreting tissue, namely, the mucous membrane, formation of inelastic bands or segments which do not permit adequate closure of the outlet, damage to the voluntary muscle with varying degrees of incontinence, damage to the involuntary sphincter and incomplete healing. The early recurrence of hemorrhoids is most likely due to inadequate removal at operation.

Any presentation on hemorrhoids should carry a note of warning that adequate diagnostic procedures be employed to rule out the presence of neoplasms and other disease before treatment is instituted. This includes a complete history, a careful digital and a thorough sigmoidoscopic examination after adequate preparation of the patient. Roentgenoscopy is reserved for the study of the parts above the reach of the sigmoidoscope.

The preoperative preparation includes phthalysulfathiazole (sulfathalidine) begun about four days prior to operation in doses of 1 Gm. four to five times daily and continued for seven to ten days after operation. The use of this drug seems to be followed by fewer instances of abdominal distention and by more that have spontaneous defecations than is seen when mineral oil or bulk laxatives are used. Furthermore it may reduce the tendency to fibrosis due to low-grade postoperative infection.

A bland diet is ordered twenty-four hours preoperatively to reduce the fecal content of the lower bowel so it may be cleaned out adequately by enema the evening before operation, and also to minimize abdominal discomfort following operation.

Sedation is obtained by the administration of sodium pentobarbital gr. 1½ the evening before operation; if local or re-

gional anesthesia is employed, this drug is repeated one hour prior to the scheduled time of surgery; in addition, morphine sulfate gr. $\frac{1}{6}$ and scopolamine in an ampule gr. $\frac{1}{150}$ to $\frac{1}{200}$ is administered hypodermically one-half hour before operation.

in hypertensive patients. Other cardiovascular stimulants have never been found necessary, the blood pressure changes varying in either direction not more than 10 mm. of mercury from the pre-anesthetic reading.
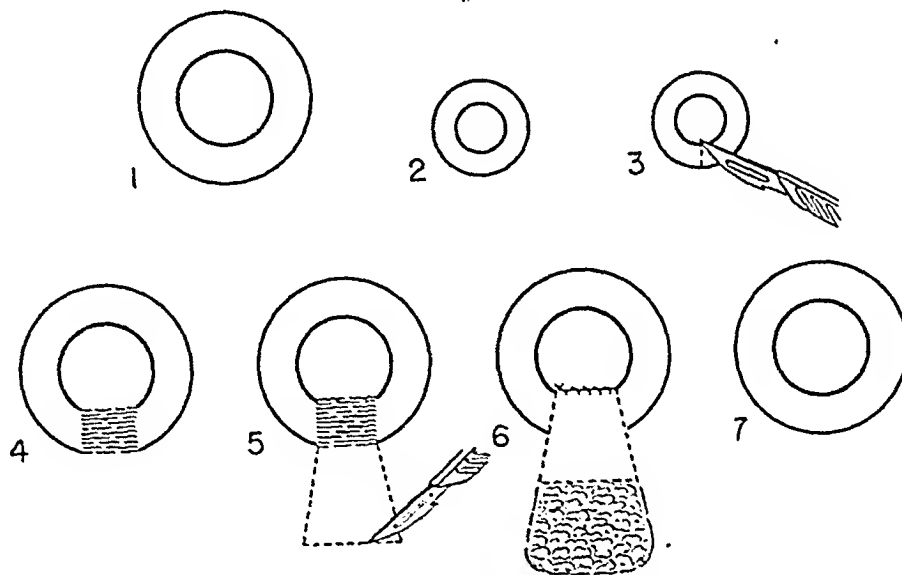
FIG. 1. Diagram illustrating preparation of sliding graft and anusotomy in plastic repair of anal stenosis, and also employed to prevent contractures following radical hemorrhoidectomies. 1, Represents normal anal lumen; 2, a stenosed canal; 3, indicates the anusotomy; 4, shows the separation of the wound edges and consequent widening of the anal tube; 5, represents the incisions for preparing the sliding skin graft; in 6, the graft has been drawn up to cover the entire surface of the anal wound except for two narrow radiating slits; a large open wound remains on the outside far from the margin; 7, indicates the end result.

Complete physical examination, urinalysis, complete blood cell count, hemoglobin and blood clotting time determination are part of the routine.

Spinal anesthesia in a much smaller dose than is customarily used has been employed by me over a period of fourteen years. Twenty mg. of procaine (1 cc. of 2 per cent solution) in a commercially prepared ampule, which also contains suprarenin 1:20,000, has been found to afford anesthesia for a period of one hour or longer. The omission of suprarenin may shorten the duration of anesthesia markedly. The small dose of procaine subarachnoidally seems to cause less strain on the patient's tissues generally and locally than other anesthetic methods and dosage heretofore used. The amount of suprarenin is so small that it apparently need not be feared

The time-honored lithotomy position has been replaced largely by the Buie prone position with buttocks elevated, and by either the left or right lateral position.

Reagents which produce prolonged anesthesia definitely have been established to result in less postoperative pain, which is evidenced not only by the smaller number of doses of the narcotic employed but also by the calmer attitude of the patient. The oil-soluble reagents and diothane solution 0.5 per cent have been found to be satisfactory. Yet even more important than these in the reduction of postoperative pain is judicious, adequate and gentle surgery for which there is no substitute.

The objectives mentioned at the outset will be secured if the surgeon restores or retains the anatomic features, namely, a canaly of sufficient caliber to admit readily
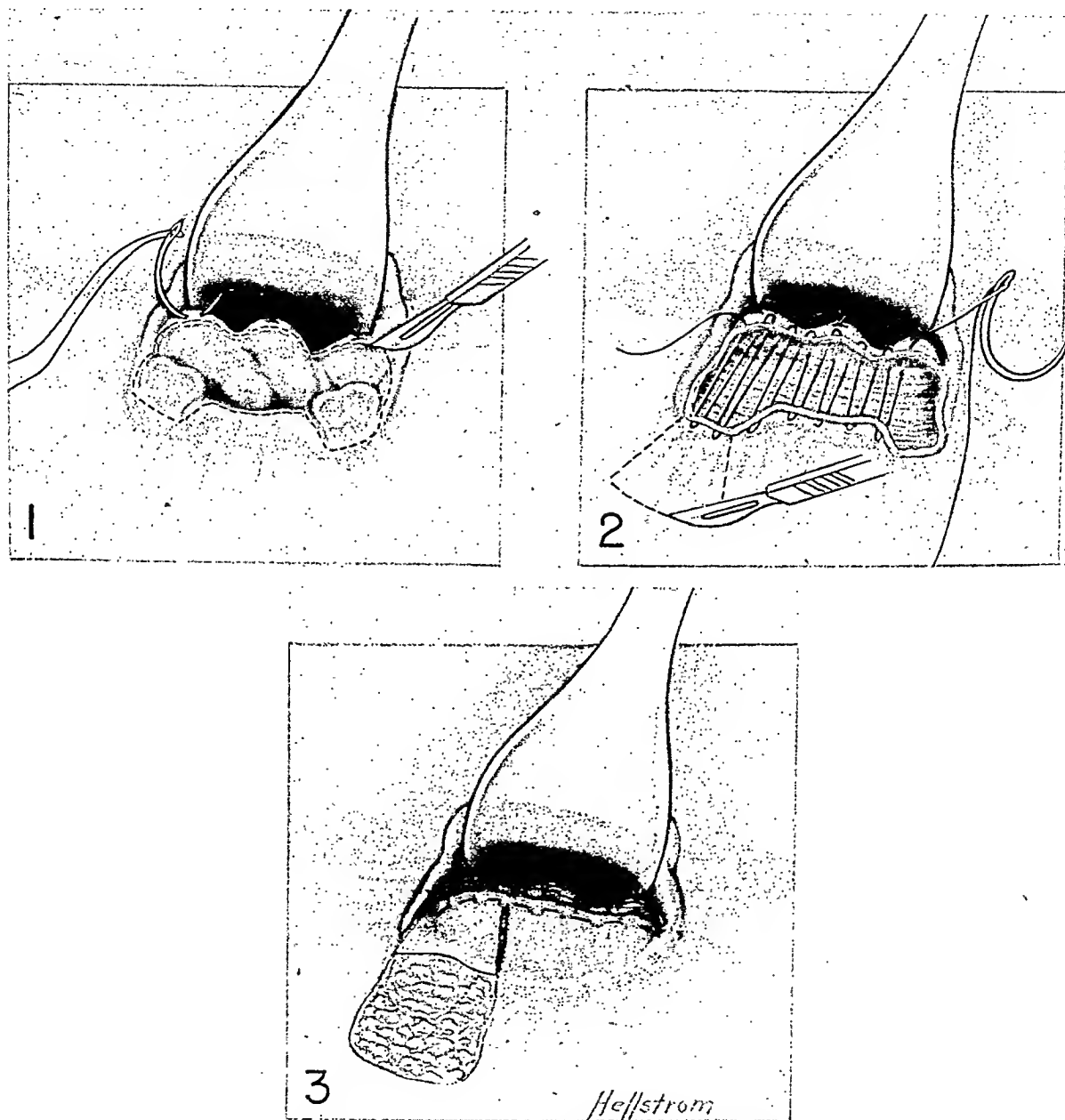
FIG. 2. Radical type of circumferential hemorrhoidectomy, with utilization of sliding graft. In 1, three initial transfixion sutures are shown; the dotted lines indicate the lines of excision on one hemicircumference; 2, depicts the outline of a sliding graft and the sutures employed for loosely coapting the wound edges; in 3, the graft has been drawn up into the anal canal, and the suturing completed; the wound edges in the right side of the illustration could easily be united without producing tension, hence no additional graft is shown.

at least two fingers with the patient under anesthesia, walls that are elastic, distensible and contractile and a mucocutaneous junction which is no lower than the normal pectinate line, that is about 2 to 3 cm. from the anal margin.

In general, two surgical methods are now in use, namely, the radial excision method and the circumferential excision or amputation method.

Radial excision procedures are apparently the most frequently employed and it has been emphasized—and rightly so—that adequate islands of uninjured tissue should be left lest stenosis ensue. Many such technics have been described which are

Carmel—Hemorrhoids

satisfactory for patients with simple cases involving scarcely more than the three classical sites of the tube, namely, the left lateral, the right posterior and the right anterior segments.

The patients with extensive cases still posed a problem for the surgeon studying anorectal diseases. If all the involved tissues are simply removed either radially or by amputation and suture of the wounds, the undesirable sequelae mentioned are prone to follow. On the other hand, if all the abnormal tissue is not removed, as has been frequently advocated in order to avoid sequelae, the result again is apt to be unsatisfactory, in that the patient's symptoms may not be eradicated completely or early recurrence may ensue.

Contributions toward the solution of the dilemma include the creation of anal flaps in surgery of prolapsed strangulated hemorrhoids,[1] suturing of the superior cut edge of the mucous membrane to the proximal fibers of the external sphincter muscle[2,3] and creation of three or four isthmuses by suturing the cut mucous membrane to the corresponding cut skin and employing available external tags to aid in forming the isthmus while allowing the rest of the wounds to granulate.[4]

It is generally conceded that tissues sutured properly without tension heal more satisfactorily than raw granulating surfaces. I have found it quite advantageous to employ sliding skin grafts and relaxation incisions and occasionally anusotomy to aid in filling defects of the anal canal and to prevent contractures. The end result tends to be a tube which simulates the normal hitherto unoperated one. The method was first used in 1936 in correcting a marked anal stenosis (Fig. 1), in patient H. L. at Longview Hospital, and was orally described by me at a round table discussion at a meeting of the Ohio State Medical Association in Cincinnati, 1940.

After the hemorrhoidal tissues have been removed a graft is prepared by making an incision in the perianal skin about 1.5 to 2 cm. lateral to one or more of the wounds made by the dissection of the external hemorrhoids. At each end of this incision a radiating cut is made, thus creating a segment of skin detached at all its edges and attached only by its deep surface and capable of being slid without tension well up into the anal canal to the level of the cut edge of the mucous membrane. (Fig. 2.) The medial edge of this skin graft is sutured to the superior portion of the external sphincter muscle and to the mucous membrane which is not pulled down but rather the skin is pulled up to it. Thus, the anal canal becomes relined by tissue of ectodermal origin and the mucocutaneous border is reestablished at the proper level. The radiating cuts not only help to free the sliding graft but serve as drainage slits. Sometimes it is found that the incision which is made lateral to the anal margin releases the tension sufficiently to allow the skin to be pulled up to cover the defect completely, making it unnecessary to add the radiating cuts; this constitutes a relaxation incision. One or two—rarely three—such incisions or grafts are employed.

The addition of relaxation incisions and sliding grafts to any method of hemorrhoidectomy permits the removal of all diseased tissue and the final results of such a plastic repair frequently challenge detection of any operation having been performed or of disease having existed.

COMMENTS AND SUMMARY

1. The objective of modern hemorrhoidectomy is the complete restoration to normal by the removal of all diseased tissue and the application of plastic methods of repair when indicated.

2. Before treatment is instituted the presence of neoplasms and other disease is ruled out by a complete proctologic examination including a thorough sigmoidoscopy; x-ray is employed for the detection of lesions above the level that can be reached by the sigmoidoscope.

3. Phthalysulfathiazole (sulfathalidine) seems to add to the postoperative comfort of the patient by lessening abdominal dis-

tention and by promoting spontaneous defecations.

4. Spinal anesthesia, with a much smaller dose of anesthetic reagent than is customarily used, is advocated. One cc. of a 2 per cent procaine solution containing suprarenin 1:20,000 (20 mg. of procaine) has been found to be effective and innocuous in both hypertensive and hypotensive patients. The use of suprarenin in the procaine solution doubles or may even quadruple the duration of anesthesia.

5. For the reduction of postoperative pain the most important factor is good surgery; reagents which produce prolonged anesthesia are of value.

6. True sliding grafts and relaxation incisions in conjunction with hemorrhoidectomy are described. Their use permits radical removal of hemorrhoids and restoration to normal without sequelae.

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DISCUSSION

HUGH BEATON (Fort Worth, Tex.): Someone has said, "The most successful man is the man who holds onto the old just so long as it is good, and grabs the new just as soon as it is better." Dr. Carmel has done that. Certainly we all agree with him about the value of a thorough and complete physical examination. Embarrassing situations can be avoided by knowing the patient's physical condition before starting an operation. I have not used sulfathaladine in preoperative care. I think I probably will in some of my patients afterwards. My patients are in the hospital the afternoon before the morning of operation, in ample time for routine laboratory work followed by a good night's rest.

For anesthesia I prefer caudal, metycaine 25 to 30 cc. However, at times nitrous oxide and ether are used; also, spinal, 50 mg. (I have not been able to get along with less.) In the aged, I find local infiltration very satisfactory. In some cases I use oil-soluble anesthetics but it is my impression that they are not without danger and I believe they retard healing in some patients.

Regarding surgical technic so ably described by Dr. Carmel, I can see the work of Drs. Buie, Bacon, Fansler, Hirschman, Martin, Rosser, etc.

I have done many plastic type operations and, although using careful technic and postoperative care, every now and then I get an anal stricture which has to be taken care of in the office. I would like the Dr. to tell us if he has the same difficulty.

What we are striving for in rectal surgery is to remove all pathology, avoid unnecessary trauma and leave a clean, flat wound that heals readily. When the first dressing is removed the, anus should be free from edema and tags. At the close of operation I apply mercurochrome and a small wick of oxycel held by a T-binder.

Patients are allowed out of bed to void and are given sitz baths the following day. I do not apply warm packs unless the patient has special nurses. I keep my patients in the hospital on an average of seven days. They come to the office once a week generally for four weeks. By that time the wounds are healed; the anal canal is normal, at least it is normal so the patient can have a well formed bowel movement without pain.

I wish to thank Dr. Carmel for the privilege of discussing his paper.

A. GERSON CARMEL (closing): I want to thank Dr. Beaton for his remarks. The application of the principles and procedures I mentioned affords the experienced proctologic surgeon an increased latitude in excising all pathologic tissues.

SURGICAL TREATMENT OF CHRONIC ULCERATIVE COLITIS

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THOSE who have been following the management of chronic ulcerative colitis over the past decade or longer are keenly aware of the controversial nature of this disease.

In reviewing the records and ultimate outcome in 110 patients with chronic ulcerative colitis seen in the twelve-year period from 1935 to 1946 I am favorably impressed by those who have been classified as mild. They have continued in fair health in their respective occupations and satisfactory management has been evolved by their physician.

However, I am concerned with a group that has been classified as advanced cases of chronic ulcerative colitis. It is probable that about 15 to 20 per cent of all patients with chronic ulcerative colitis reach an advanced stage. Whether this follows an acute fulminating onset or is a result of recurrent attacks is of some importance and will be considered later.

If there is some degree of accuracy in the estimate that 15 to 20 per cent of all patients with chronic ulcerative colitis reach an advanced stage, we are then ready to discuss treatment of this group of patients.

If we are to evaluate properly both medical and surgical treatment of this group of desperately ill patients, some rules of conduct should be formulated.

It would seem wise to state that the objective of medical and surgical treatment should be "a restoration to health and previous occupation." This should also be the yardstick by which we measure the results of both methods of treatment. Finally, if we have failed to rehabilitate the patient so that a "medical derelict" or a "surgical invalid" represents the outcome of our medical or surgical efforts, we should re-evaluate our methods of treatment.

DIAGNOSTIC CONSIDERATIONS

Our patients have been classified as having chronic ulcerative colitis when they presented clinical, roentgen and proctoscopic features of this disease as described by Rankin, Bargen and Buie.[1] Amebic colitis, tuberculous colitis, bacillary and other forms of colitis have been excluded by appropriate investigation.

It seems desirable to state that a definition of an advanced stage of ulcerative colitis will be found under each surgical heading in another section. However, it is to be noted that the advanced stage of ulcerative colitis was reached by some of these patients in two rather distinct ways: (1) As a result of an initial fulminating onset, some patients arrived at a rather severe state of constitutional decline and structural change in the colon. These patients all presented intricate problems in the management of their sepsis, anemia, malnutrition and diarrhea. I have not been impressed by the results of those who advocate ileostomy as a life saving procedure for patients in this phase of the disease. Whether or not these patients die under medical or surgical auspices is dependant upon who is taking care of them at the time. (2) Those patients who, as a result of chronic or recurrent ulcerative colitis, reached an advanced stage of constitutional and visceral degenerative changes, with or without complications, were easier to manage. A few of these patients died as a result of surgical treatment aimed at restoring them to health and previous occupation but the majority survived and were rehabilitated. I am impressed by the surgical rehabilitation of this group.

When any patient became a candidate for surgery as previously described, we then

classified the indications for surgery under the terms A—Specific and B—Elective. In so doing, the reports of Cave, Jones, Rankin, Cattell, Dixon, Bargen, Bockus, Crohn, Garlock, Kiefer and many others were followed with interest and profit.

SPECIFIC INDICATIONS FOR SURGERY

Chronic Ulcerative Colitis with Constitutional and Visceral Degenerative · Changes. The typical patient in this group has had either a chronic or intermittant ulcerative colitis for several months or years. The clinical features of chronic sepsis, toxicity, anemia, hypoproteinemia, loss of weight and strength and signs of multiple vitamin deficiencies will be present. In young women cessation of menses, failure of breast development and other secondary sex characteristics may be noted. In both sexes there have been numerous prolonged periods of absence from school or work. The patient is a chronic invalid and a candidate for surgery.

Anorectal Complications. With the development of stricture, recurrent abscesses, anorectal, pelvic and rectovaginal fistula, incontinence and painful disabling perirectal infection, an insurmountable barrier to recovery has been reached. Adequate drainage of abscesses should be done but beyond this further local surgical treatment is usually unsatisfactory. The disability suffered by the majority of patients in this group prevents them from enjoying good health or full employment. They are candidates for surgery.

Polypoid Degeneration and Carcinoma. There is almost universal agreement that one should consider malignant change as the usual outcome of polyp formation, regardless of whether these polyps are considered inflammatory pseudopolyps or true adenomas. Polypoid degeneration ordinarily represents a complication of long standing chronic ulcerative colitis and diagnosis can be made by proctoscopy and careful roentgen examination. Good surgical judgment indicates that these patients are candidates for operation despite the fact that disability may be minimal in some instances.

Obstruction and Tumor Mass. An abdominal or abdominopelvic mass arising from any portion of the colon may produce obstruction. The diagnostic problem of determining whether the tumor mass is inflammatory or malignant can rarely be solved without exploration. A previous roentgen or proctoscopic diagnosis of polypoid degeneration may favor the diagnosis of malignancy. Relief of the obstruction by appropriate surgery is indicated preliminary to surgical treatment of the mass itself.

Subacute Perforation, Abscess and Fistula. With the development of an abscess resulting from perforation it is advisable to perform drainage after localization has occurred. Surgical experience indicates that the improved condition of the patient following such drainage enhances the success of subsequent surgical procedures. With the development of a fistula ileostomy or proximal colostomy should be done several weeks or months before any resection of the involved portion of the bowel is attempted.

ELECTIVE INDICATION FOR SURGERY

Focal Infection. Some individuals, as a part of their chronic ulcerative colitis picture, develop a disabling polyarthritis. It is conceded that they may be benefited by resection if the arthritis has not advanced to the stage of marked structural change in the joints, tendons, synovia, etc. Pyodermia, recurrent skin ulcers, furunculosis, neuritis, stomatitis and erythema nodosum are some of the manifestations of this disease that may be avoided or benefited by eliminating the source of infection.

Hemorrhage. I do not find myself in accord with the heroic viewpoint that chronic blood loss from recurrent or continuous hemorrhage is an indication for ileostomy. Massive hemorrhage has responded to repeated transfusions of blood, plasma and protein solutions. Massive parenteral doses of vitamin K, C, niacin, B complex and liver are part of the sup-

portive treatment. If the patient can be carried over this critical stage, a high surgical mortality rate will be avoided and chances of a favorable outcome will be increased.

It is acknowledged that some patients will die from exsanguinating hemorrhage and infection because they neither respond to supportive treatment nor become acceptable for surgical intervention.

Acute Fulminating Ulcerative Colitis. Surgical treatment should be avoided in the majority of patients who develop initial acute fulminating ulcerative colitis. An initial fulminating attack of ulcerative colitis developing in a patient who has heretofore been well should be considered a medical problem. These patients develop an advanced structural pathologic change in their colon very rapidly and the severe state of constitutional decline is quite alarming. If these patients can be carried over their initial fulminating attack, some will enter a relatively afebrile ambulatory phase and respond to medical management.

However, if they show signs of returning to a toxic febrile phase when they have apparently recovered from their initial fulminating attack, surgical intervention is advocated.

Acute Perforation. With rare exception perforation into the free peritoneal cavity during the initial acute stage of ulcerative colitis is fatal. A section of necrotic colon full of holes and the inability of the adjacent bowel, mesentery and viscera to cope with such a severe spreading peritonitis as may be demonstrated at the autopsy table is ample evidence of the futility of treatment.

SURGICAL PROCEDURE

A few general remarks concerning the object of surgical treatment are in order. If we remember that our goal is surgical rehabilitation of the patient, we must define this as a return to previous occupation and good health without limitation of activities. If we are to rehabilitate the patient, morbidity and mortality should be kept to a minimum or we have not fulfilled

the object of surgical treatment. Finally, if we have succeeded in rehabilitating the majority of these patients, our mission has been fulfilled.

The surgical procedures have been planned to suit the requirements of the patient.

Ileostomy without Colectomy. Ileostomy without colectomy was performed for six patients. Four have been restored to health. One patient died the fifth postoperative day and must be considered in the postoperative mortality figures. One patient survived ileostomy but subsequent to his discharge home developed an ileocolic fistula and died as a result. This death should be attributed to the disease and should not be considered in the operative mortality figures.

Ileostomy—Total Colectomy—Rectum Left in. Two patients have had a total colectomy performed in one stage. Ileostomy had preceded the colectomy and the rectum was left in. In one patient the rectum became involved after total colectomy was performed and in the other the rectum was involved at the time colectomy was performed. Both will have an abdominoperineal resection at a later date although their restoration to health has been accomplished. The third patient had an unsuccessful ileosigmoidostomy converted into a permanent ileostomy due to recurrent anorectal abscess-fistulas. He is employed full time.

Ileostomy—Total Colectomy—Abdominoperineal Resection. Three patients have had ileostomy, total colectomy and abdominoperineal resection of the rectum done in stages. All have survived and are gainfully employed without loss of time or disability.

Ileostomy—Right Colectomy. One patient had ileostomy and right colectomy done at the same time. She died the tenth postoperative day from perforation of the colon.

Left Colectomy—Abdominoperineal Resection. Four patients who had an ulcerative colitis involving only the rectum and

descending colon have been restored to their previous occupations. They have a terminal colostomy of the right transverse colon and are not disabled.

Ileosigmoidostomy—Total Colectomy. One patient had a successful ileosigmoidostomy followed by total colectomy performed in two stages. During her convalescent period at home she developed symptoms of incomplete obstruction due to adhesions. She was re-admitted and died following exploratory laparotomy. Her death is considered a postoperative hospital mortality in the statistics.

Segmental Resection—Anastomosis. One patient had resection of the descending colon for a segmental form of chronic ulcerative colitis. End-to-end anastomosis between the transverse and sigmoid colon was performed. She is gainfully employed.

Colostomy—Inoperable Carcinoma. One patient had a colostomy performed in the right transverse colon to relieve obstruction. This patient subsequently died in the hospital from carcinoma of the sigmoid arising in a segment of ulcerative colitis. Her death is correctly attributed to the disease and not to the operative procedure.

ANALYSIS OF SURGERY

It is difficult to analyze the status of the operative procedures for in tabulating these it was found that twenty patients underwent thirty-five operations. A postoperative mortality of 8.5 per cent due to the operative procedures is acknowledged as correct. Following operative procedures a mortality of 5.7 per cent due to the disease, but not the operative procedure, is likewise correct.

In commenting on Table I it is to be noted that under numerals II, III and V the rather formidable procedures of subtotal or total colectomy with or without abdominoperineal resection of the rectum were done without a fatality. This is indeed a fortunate and happy experience for I have developed a distinct feeling that if I can get my patients over the ileostomy

stage of their operation the rest would be easier.

In commenting on Table II a restoration to health and previous occupation was accomplished in 70 per cent of patients. This is indeed gratifying for it should be

TABLE I

STATUS OF OPERATIVE PROCEDURE IN TWENTY PATIENTS

35	Operative Procedures	Deaths	
A	Postoperative Mortality	No.	Per Cent
I	6 Ileostomies without colectomy	1	16.6
II	3 Ileostomies—total colectomy—rectum left in	0	
III	3 Ileostomies—total colectomy—abdominoperineal	0	
IV	1 Ileostomy—rt. colectomy	1	100
V	4 Left colectomy—abdominoperineal	0	
VI	1 Total colectomy—ileosigmoidostomy	1	100
VII	1 Segmental resection—anastomosis	0	
		3	8.5
B	Chronic Ulcerative Colitis Mortality		
	1 Ileostomy—ileocolic fistula (from 1 above)	1	100
	1 Colostomy—carcinoma—inoperable	1	100
		2	5.7

TABLE II

STATUS IN RELATION TO OBJECTIVE IN TWENTY PATIENTS

		Per Cent	
12	Restored to health	60	70
2	Convalescent	10	
3	Postoperative mortality	15	25
2	Chronic ulcerative colitis	10	
1	Unknown	5	5
20		100	

obvious that rehabilitation of such a seriously ill group of individuals is a great problem.

In commenting on Table III I would like to point out that a direct relationship be-

tween operative procedure and cause of death is present in three patients. However, in two patients death could not be attributed to operative intervention by any stretch of the imagination. One patient formed an ileocolic fistula during his con-

TABLE III

MORTALITY STATUS IN FIVE PATIENTS

A	Postoperative Mortality	15 per cent
1	Ileostomy—perf. ilium—peritonitis	
1	Ileostomy—rt. colectomy—perf. colon—peritonitis	
1	Explore—adhesions—peritonitis	
B	Chronic Ulcerative Colitis Mortality	10 per cent
1	Ileostomy—ileocolic fistula	
1	Colostomy—carcinoma—inoperable	

valescent period. This effectively voided the previous ileostomy and his death is correctly attributed to an active resumption of ulcerative colitis. The other patient died from carcinoma of the sigmoid and colostomy was performed only as a palliative procedure.

SUMMARY

1. In our limited experience it is believed that approximately 15 to 20 per cent of patients who have chronic ulcerative colitis will become candidates for surgery.

2. These patients will be advanced cases of ulcerative colitis as a result of either an acute fulminating phase or a chronic intermittant process.

3. A group of patients with advanced chronic ulcerative colitis were classified as candidates for surgical treatment under two headings: A—Specific and B—Elective.

4. Operative procedures have been briefly outlined.

5. A restoration to health and previous occupation, the objective of surgical treatment, was accomplished for 70 per cent of the patients.

DISCUSSION

ROBERT A. SCARBOROUGH (San Francisco, Calif.): Dr. Ault's paper, to which you have just listened, is a splendid exemplification of the type of critical analysis of one's own experience which all of us should periodically employ for our own good and for the good of our patients.

Dr. Ault has reason to be pleased and satisfied with the results of his surgical management of this disease. He has expressed no such pleasure or satisfaction, however, but rather encourages discussion of his failures.

There were five deaths in twenty patients. There was one death following ileostomy. This much comment is made only to emphasize that the performance of ileostomy for chronic ulcerative colitis is a serious major surgical procedure which can be followed by extremely high mortality unless the time of operation is wisely selected and unless preoperative and postoperative care are suitably controlled. Dr. Ault's mortality rate following ileostomy, which is actually $8\frac{1}{3}$ per cent and not $16\frac{2}{3}$ per cent, as shown in his own tables, is well below the average mortality rate for this procedure.

One patient died following simultaneous ileostomy and right colectomy. We believe that ileostomy alone should be a first stage procedure prior to any subsequent resection for chronic ulcerative colitis.

One patient died of peritonitis following ileosigmoidostomy and colectomy. This case would appear to exemplify the hazards of complications by infection in the performance of anastomosis in a colon involved by this disease.

The fourth death followed the development of an ileocolic fistula subsequent to ileostomy. This was an unforeseen and unavoidable complication which might have been prevented by early colectomy if it could have been anticipated.

The final death from inoperable carcinoma superimposed on chronic ulcerative colitis is of significance as an example of the potentiality for malignant degeneration in this disease. The time to cure the patient had passed when he first came to Dr. Ault. Only colectomy before malignancy developed would have saved this individual.

In our own experience, one of the most difficult decisions to be made has been that of just when to perform ileostomy. I agree with

Dr. Ault that it is not indicated in acute fulminating ulcerative colitis or for the treatment of hemorrhage.

It is indicated in those cases of chronic disabling disease which have failed to respond satisfactorily to medical treatment.

Subsequent colectomy should be employed when septicemic toxic effects persist or when there is evidence of the development of polypoid mucosal proliferation, since this is a definite precursor to malignant degeneration.

VERNON G. JEURINK (Denver, Colo.): Not many years ago, we frequently heard the statement made that chronic ulcerative colitis was never a surgical disease, even when complicated by various conditions which now almost universally are considered indications for surgical interference.

Dr. Ault's presentation and Dr. Scarborough's discussion should impress us, along with an increasing volume of similar reports by other workers in this field, that surgery has a great deal to offer in the selected cases of chronic ulcerative colitis. Because of our personal inclination to leave well enough alone, we have been inclined to let our patients go along with ileostomy so long as their condition remains stationary or improved. When these conditions do not exist, or when stenosis, polypoid change and related changes occur, we recommend colectomy.

Recently, in ten cases only we have tried a new drug called nisulfazole which is still in the investigative phase. This is obviously not a sufficient number of cases from which to draw any worth while conclusions; but it is our impression that it may prove to be of some value in quieting acute exacerbation of the condition.

It has been a pleasure to hear Dr. Ault's paper and Dr. Scarborough's discussion.

THOMAS T. MACKIE (Winston-Salem, N. C.): It is a privilege to have the opportunity of discussing a paper of this sort. I, of course, am not a surgeon and I must confess that I am cheered to find a surgeon who has such a conservative point of view with respect to this very distressing disease. I agree completely with Dr. Ault's philosophy. I would add only one additional indication in his specific group and that is the rare case in which the extensive pathological development of this disease is restricted to the proximal colon. In those instances, I believe one should consider surgery at a very much earlier date than we do in the other types.

I agree completely that surgical intervention in the elective group is almost invariably followed by serious results and very seldom fulfills the objective which Dr. Ault has defined as the purpose of surgery, namely, the restoration of the individual to health and to a productive life. He has brought out also by indirection one of the other extremely important aspects of this problem which has impressed me over the time that I have been studying it. That is that there are very few of us who have sufficient breadth of experience to be fully competent to evaluate all of the problems which the individual case will present and that in effect many of these patients, and particularly the seriously ill ones, are conjointly medical and surgical problems, and that the best results occur when the internist and the surgeon are working together.

Again, I should like to congratulate Dr. Ault on a very interesting paper and, obviously, on very skillful surgery.

FRANK C. YEOMANS (Great Neck, N. Y.): I have no prepared discussion but I want to express my appreciation to the essayist and at the same time to emphasize again, of course, that chronic ulcerative colitis is essentially a medical disease. It stays medical until pathologic complications supervene. I have seen a number of cases of ileostomy and most of those patients are far from happy.

I want to call attention again to a surgical procedure that is now very much neglected. It was used a long time ago in cases of chronic ulcerative colitis that do not respond to treatment and are not complicated, we will say, by polypi. Appendicostomy is a simple and safe procedure and may result in great satisfaction.

At the meeting of the New York State Society in 1946, I showed a patient and reported on a man—I think he was fifty-six years of age—who was in a very desperate condition with chronic ulcerative colitis. He had been ill for months and received all the surgical treatments, including chemotherapy, transfusions and all that in a very good hospital and under a man who is very competent and has a large experience. He was given up by the doctor and told that he could either stay in the hospital and die or go home and die. So he was brought to the Polyclinic Hospital in an ambulance. He weighed about 90 pounds, had a temperature of 103°F., and was anemic,

lethargic, and seemed to be a candidate for the mortuary.

We tried some more medical treatment which failed. Then I did an appendicostomy, and through this opening irrigated the colon and instilled sulfa drug in suspension. In three days the picture changed. The ulceration healed completely and the man has remained well since a year ago last October. He still has the appendicostomy.

The advantage of appendicostomy is that if you have to do major surgery later, the appendecostomy does not interfere with it at all. It may itself be the life-saving procedure. If it is properly done, only a very small catheter, say a No. 12 French, is inserted and removed after each treatment. The patient learns to pass it himself. It is no inconvenience at all. There is no bag worn and it has all the advantages of a simple thing. It must not be closed. The mucosa grows to the skin; the stoma does not contract. It should not be closed because you do not know when it may be needed at a subsequent time. I have had patients who have had the appendicostomy for years and have had to use it on various occasions.

I bring that up simply to show that that procedure can be performed under local anesthesia or a little pentothal. It is not a major procedure comparatively and I bring it to your attention as a very useful and at times a life-saving procedure of a minor nature compared to the other.

GARNET W. AULT (closing): As I left the platform, several people asked the name of that bag. I will put it on the blackboard for you— *Rutzen*. I will have two of these bags on the table after the meeting so that you can examine them.

I wish to thank all discussers for their pleasant comments and am delighted to know we have some agreement and some very critical analysis. I would like to reiterate the fact that colostomy or ileosigmoidostomy should not be done in the presence of an involved segment of the bowel. I have not done that and I purposely made that plain and pointed out in one of those illustrations that the involved

rectum would not stand the anastomosis of the ileum to it.

Dr. Scarborough has very definitely brought out the fact that polypoid degeneration will lead to carcinoma, and when your patient has reached that stage he is not a candidate for anything. We recognize that in the one patient in whom we did ileostomy, (as a part of a right colectomy) an error of surgical judgment was made. That is acknowledged.

I want to thank Dr. Jeurink for his discussion. We use the sulfas, streptomycin, penicillin and all other forms of chemotherapy as useful adjuvants.

I am delighted to hear that Dr. Mackie could be here and I appreciate his discussion. I am in thorough agreement that with lesions involving the proximal colon, we should do our surgery earlier. I am in complete agreement that these patients should have joint medical and surgical management. If you attempt to manage these patients alone from the surgical standpoint, you are headed for trouble. You should have joint management and you will have much better results.

I do wish to point out that 15 to 20 per cent of all cases of ulcerative colitis become problem cases. Whether they become problem cases due to an initial acute fulminating attack or due to chronic recurrent attacks is an entirely different thing; both require joint management. In the group of 15 to 20 per cent, who become difficult to manage, surgery has something to offer these patients who can be considered candidates for surgery.

I want to thank Dr. Yeoman's for his discussion. I would like to ask why are Dr. Yeomans' patients not happy with ileostomy? I am wondering if some people are not still using apparatuses that are not suitable for ileostomy. The Rutzen bag is the best answer to ileostomy. It is the only thing that we know that has made surgery acceptable to the physician and to the patients. If you are going to do an ileostomy and put an ill-fitting bag on, put sea sponges or something else on the surface, you are not going to have a happy patient and you are not going to have a very happy doctor very long, either.

CHRONIC DIARRHEAS*

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CHRONIC diarrhea is perhaps the most grossly mismanaged condition in all of medicine. The reasons are not far to seek: The first is that it is likely to be treated by home measures or drug-store advice until it has become established and until in some patients it is no longer simple. The second reason is clear from the first. It is regarded as a clinical entity and is treated as if it were whereas actually it is a symptom for which an underlying cause must be sought. At best the etiologic diagnosis is not easy, in that it is a laboratory matter which calls for special technics. At worst it is long and tedious and often unrewarding. There are, however, no short cuts and it is the attempt to take them which leads to so many difficulties in the diagnosis and treatment of diarrheal diseases.

The diarrheal diseases are with rare exceptions non-lethal but they represent an important problem in medicine today. They have a high incidence. They affect all groups and classes although their frequency increases as one descends the social and economic scale, their spread being favored by poor hygienic habits, which unfortunately go hand in hand with poverty. Their spread is also favored by the fact that many patients affected with them do not consult physicians. In their extensive survey of acute diarrheal diseases, for instance, Hardy and Watt found fecal cultures positive for shigella in 380 persons, of whom only two were under the care of a physician; one of the two died three days later. As these writers note, in the absence of their special study, there would have been 378 undetected infections and a corresponding unrecognized number of hidden sources of new infections.

Another reason for the spread of diarrheal disease is the frequent failure of the physician who is consulted to take the time and make the effort required to identify the etiologic agent. It is unfortunate that diarrhea usually responds rapidly, although often transiently, to simple measures. Under the circumstances it takes a certain strength of character on the part of both physician and patient to undertake a long routine of investigation.

Definition. A discussion of the chronic diarrheas properly begins with a definition of terms. Diarrhea must be distinguished from dysentery. The etiologic bases of the two symptoms are sometimes different and from the standpoint of severity they are always very far apart.

It is both useful and convenient to make the distinction in terms from the patient's point of view. The passage of unformed stools characterizes both diarrhea and dysentery. Diarrheic and dysenteric stools may both contain mucus, pus and blood but a dysenteric stool also contains cellular débris, it contains little fecal matter or none at all and its passage is always associated with tenesmus. A diarrheic stool, on the other hand, whether the diarrhea is acute or chronic, is chiefly fecal in composition.

From the patient's point of view diarrhea represents an abnormality in bowel habit as compared with the usual normal habit. It may manifest itself by an increase in the number of stools as well as by a change in their characteristics. What would be diarrhea to one person would not be at all ab-

normal to a person whose normal habit was to pass two or more semiformed or soft stools daily. Detailed questioning, however, is always necessary for definitions of normal differ. One patient in the writer's experience who stated that her bowel function was normal, later revealed that for years she had had as many as eight bowel movements daily. A dysenteric stool, on the other hand, is not a matter for relative consideration. It would be considered abnormal by any patient, regardless of what his usual bowel habits were.

The classification of diarrhea into acute and chronic has nothing to do with the severity of symptoms but is entirely related to time. For the purposes of this discussion a chronic diarrhea is any diarrhea which is continuous or recurrent and which has lasted longer than a month.

ETIOLOGIC CONSIDERATIONS

On the basis of personal practice, which for the past eleven years has been confined exclusively to diseases of the colon, it may be said that amebiasis, shigellosis and brucellosis account for two-thirds to three-quarters of all patients with chronic diarrhea who are seen. Whether this is generally true is perhaps open to debate. The writer's practice is chiefly consultative and much of it comes from outside of Louisiana. It is doubtful, however, that his practice represents an accurate cross section of the diarrheal diseases in the United States and it is equally doubtful, in spite of a large number of patients referred from Central America, whether it represents an accurate cross section of these diseases in the tropics.

In the order of frequency, amebiasis is the most common cause of chronic diarrhea although if the physician relies upon diarrhea alone to make the diagnosis, he will miss more than one-half of all his patients. This is a disease in which constipation is an important part of the clinical picture.

The second most frequent cause of chronic diarrhea is shigellosis. In the writer's experience it far exceeds the num-

ber of patients with bacillary dysentery observed over a comparable period of time. Not more than 20 to 30 per cent of all patients with shigellosis, furthermore, can supply a history of previous dysentery although a carefully taken history very often reveals previous abdominal complaints. They may be little more than vague discomfort but they justify the assumption that one is dealing with a long-standing low grade infection.

Brucellosis, although its importance as a major epidemiologic problem in the United States is becoming clear, is still not frequently recognized in its intestinal form. In twenty-six patients recently seen in consultation, it had previously been suspected as the explanation of chronic diarrhea in only two instances. The other diagnoses included amebiasis, amebic dysentery, malaria, spastic and non-specific ulcerative colitis, cholecystitis and diarrhea of undetermined origin. The number of recognized cases of intestinal brucellosis is increasing probably because the disease is being sought for. The writer's experience is that effect. Over a five-year-period when no particular endeavor was being made to identify the disease, only nine cases were observed. The twenty-six patients mentioned were seen over a two and one-half-year-period during which the possibility of intestinal brucellosis was increasingly borne in mind. At the present time six patients with this disease are identified every month.

Another point to be emphasized in consideration of the chronic diarrheas is the possible coexistence of two or more different types of intestinal infection in the same subject. It has been known for some time that amebiasis can coexist with brucellosis but personal experiences with returning servicemen from the Southwest have been particularly striking in this respect. Some of them have presented extremely obstinate amebic infections which did not respond to treatment based on sound concepts and which were recognizable as...

vestigation revealed the reason, that a Shigella infection was present also, and therapeutic results were not achieved in amebiasis until shigellosis had been controlled with autogenous vaccine.

A similar situation may develop when amebiasis coexists with brucellosis. When it does, the amebiasis will be refractive even to adequate treatment until treatment for brucellosis is carried out. Indeed, refractive amebiasis, after adequate treatment, is sufficient indication for a search for brucellosis whether or not the disease is associated with intestinal manifestations. The writer's own practice is highly selective but it is probably suggestive of the general clinical picture so far as chronic diarrheas with dual etiologies are concerned.

With the exception of passing mention of one or two puzzling but relatively unimportant diarrheal diseases, this discussion will be confined to the three most common ones, amebiasis, shigellosis and brucellosis, and they will be discussed only from the standpoint of certain considerations which in the writer's experience have proved to be important. There will be no discussion diarrhea due to giardiasis, which is not common. Schistosomiasis is not of significance in the United States and strongyloi-These, while it is important, is not a common cause of diarrhea. There will also increase discussion of such conditions as the econoneas associated with lymphopathia by poeum or with chronic intussusception, nately th new growths in which roentspread gic and sigmoidoscopic examinations many establish the diagnosis. A malignant consult pist always be in the background of vey of acician's consciousness, especially in Hardy as over forty years of age, when they tive for ost commonly. It is a sound rule to only two a digital and a sigmoidoscopic cian; onation and to have a barium enema As thesced promptly in any patient in their specood appears in the stools and can-378 undcplained by hemorrhoids; it is too a sign of neoplastic disease to

rhea as a clinical entity and not as a symptom for which a cause must be sought.

CLINICAL PICTURE

The symptomatology of the three most important causative diseases, amebiasis, shigellosis and brucellosis, is remarkably similar. Common to all three are such symptoms as nervous irritability, easy fatigue, low grade fever, vague abdominal symptoms, arthritic manifestations, vague muscular pains and similar indefinite complaints. Both shigellosis and amebiasis may be characterized by regularly intermittent diarrhea, extending over a period of twenty-four to thirty-six hours and associated with malaise, frequently severe enough to require bed rest. If arthritic symptoms are part of the symptomatology, they are likely to be exacerbated during the diarrheal episodes, which have a notable seasonal incidence. In New Orleans exacerbations are most frequent between April 15th and June 15th and between August 15th and October 1st although why they appear at these times is impossible to explain.

Patients with disease of the colon are also likely to have undergone distinct personality changes. It might be added that their nervous status is in no wise improved by the fact that their complaints have frequently been diagnosed under a wide variety of terms and treated, without relief, by a variety of methods. One might be tempted, just as physicians previously consulted have been tempted, to discuss this variety of symptoms and syndromes under the general heading of neurosis, on the ground that they could not possibly arise from a single pathologic basis. The proof that they can, however, is two-fold: (1) the identification of the causative organisms in the stools in shigellosis and amebiasis and diagnosis by other measures in brucellosis and (2) the results secured in all three diseases in respect to symptoms by adequate treatment. This does not mean, however, that neurogenic diarrhea is not a clinical entity. The statement is fair that the larger the

number of such diagnoses in one's practice, the poorer are one's laboratory facilities. The diagnosis is practically always fallacious. A patient with chronic disease of the colon is likely to develop a chronic anxiety state against a background of chronic abdominal discomfort. But if the patient is thoroughly investigated it will usually be found that the history of colon disease is followed by the development of the chronic anxiety state and that the reverse sequence is infrequent.

Amebiasis. Amebiasis will be easier to comprehend if certain facts concerning it are borne in mind:

1. The parasite has a special predilection for two sites in the colon, the cecal and the rectosigmoidal areas, and the character of the symptomatology depends upon the location of the infectious process. Cecal infections are by far the most frequent. When they are present, amebiasis may assume the so-called syndromic form and simulate such intra-abdominal conditions as peptic ulcer or chronic appendicitis or cholecystitis. In infections in this area, constipation and not diarrhea is the outstanding symptom. When the infection is entirely or chiefly in the rectosigmoidal area, the disease takes a dysenteric or diarrheic form which may be either acute or chronic.

2. The passage of cysts is the rule in cecal amebiasis and the rather general belief that they are of no importance and that trophozoites are necessary for diagnosis (and for pathologic manifestations) is completely unjustified. In the opinion of the majority of American protozoologists, Endamoeba histolytica is a tissue parasite, not an inhabitant of the intestinal lumen. The "carrier" concept is therefore erroneous in this disease since tissue parasites are always pathologic and are never innocuous.

3. In neglected and badly managed subjects or in patients who develop lowered resistance for any reason, even subclinical amebiasis may assume a more serious form. Dysenteric symptoms may appear and may be followed by intestinal perforation and

ameboma (amebic granuloma) as well as by hepatitis and liver abscess, the most common of the extraintestinal amebiases, or by involvement of other organs and systems.

Certain manifestations of amebiasis, all in children and all completely different from those in adults, have recently come to the writer's attention. This special symptomatology had not previously been observed, probably because it had not previously been investigated from the standpoint of possible amebiasis. Over the past eight months, in consultation with Dr. Maud Loeber, seventy-three patients with these special manifestations have been seen. The age range in the group was from six months to twelve years but the great majority of the children were under six years of age, a fact worth emphasizing because it has been the general belief that amebiasis does not occur in young children. Apparently, if one looks for it, it does.

The symptoms were bizarre and equivocal. The children, for the most part, were not really sick, they were just not well. While the symptoms varied widely, four characteristics of the condition were outstanding: (1) The complexion had a peculiar muddy, yellowish tinge which sometimes suggested fading suntan; (2) the liver was enlarged and tender; (3) personality changes were notable. Ordinarily normal, happy children, as well behaved as young children on the average are, became irritable, sullen and unmanageable. They had tantrums of temper. They struck attitudes of defiance. They seemed actually to take delight in baiting their parents and nurses; (4) the appetite was capricious and sometimes insatiable.

In addition to these quite constant manifestations, there were other more inconstant symptoms such as mild nausea and vomiting, low grade fever, headaches, increased fatigability, an increased susceptibility to colds, sometimes mild abdominal pain which woke the child from sleep or kept him from play, muscular pains in the arms, chest and legs and occasional, mild convulsive seizures of no recognizable pat-

tern. Slight constipation was frequent. Diarrhea was present in a few patients and mild dysentery in a few others. All laboratory findings were normal.

One might question the validity of these symptoms and especially their attribution to a single cause, except that in all seventy-three patients the diagnosis of amebiasis was made in the laboratory and was indubitable. Moreover, in every instance in which adequate therapy was instituted, there was prompt improvement in the symptoms.

The explanation of the occurrence of the symptoms (although for their pattern there is no explanation) was quite simple: In every case the parents, or others in close contact with the children, were found to have amebiasis which usually was not suspected and which unquestionably was passed on to them. When the diagnosis and then the explanation became apparent in the first few patients of the series, suspicion was aroused and the later patients offered no diagnostic difficulties. The infections in this group of patients were evidently light for identification of the parasites in the stools was on the whole unsatisfactory. The diagnosis could usually be made without much difficulty from aspirated material secured at sigmoidoscopic examination and sigmoidoscopy therefore was made part of the diagnostic routine. Undoubtedly, vague symptoms of the sort listed have gone unexplained in children in the past because the laboratory examinations which would elucidate them were not made. In the light of this experience, it would seem that pediatricians must add to their diagnostic regimen in cases of obscure symptoms in young children examination of the stools for ameba and sigmoidoscopy.

There was nothing particularly surprising in the experience just related. As one's experience with diseases of the colon increases, one becomes more and more impressed with two clinical facts: The first is that these diseases seem able to mimic not only the symptoms but the actual syndrome of many other diseases. The second is that the presence of amebiasis in particular seems to exaggerate and exacerbate symptoms of diseases with which it coexists although this may not be realized until the amebic disease is controlled and improvement in the companion disease becomes evident, without treatment directed toward it.

Amebiasis mimicks the combination of vague and inconstant symptoms already commented on which internists, for want of a better name, call neurosis or the anxiety state. It mimicks cholecystitis, appendicitis and peptic ulcer. It mimicks pelvic disease and it aggravates such gynecologic states as dysmenorrhea and the menopausal syndrome. It confuses the obstetrician when it appears in the third trimester of pregnancy when, for some reason, it is likely to become apparent. It aggravates sinusitis. Even the dermatologist is likely to find it entering the clinical picture. It is a disease, in short, which specialists in all branches would do well to bear in mind as a diagnostic possibility.

DIAGNOSTIC CONSIDERATIONS

The etiologic diagnosis of chronic diarrhea is a laboratory procedure. Nonetheless, the first step in the diagnostic routine should be to secure an accurate history. The investigation should begin with a careful inquiry into the patient's normal pattern of intestinal function. In diseases of the colon it is not the number of stools or their character but their difference from the usual norm which establishes the existence of the symptom.

As a rule, only suggestive, not conclusive information can be secured from the history but a history which brings to light nothing at all still has a negative value. It may throw light upon a number of important points. An inquiry into the bowel habits of the remainder of the family, for instance, should never be omitted. Even when habits of hygiene are good, it is not always possible to avoid the spread of an infectious disease and the information ob-

tained about other members of the family may be more revealing than the patient's own history. Occupation, dietary habits, the source of milk ingested, food dyscrasias, the relationship between symptoms and the physical and emotional state, places of residence, military service, these and other data may all provide clues to diagnosis which will save time, effort and expense.

Laboratory Diagnosis. The laboratory diagnosis of all of these diseases is difficult. It is missed in a very large number of patients because diagnostic attempts are not persisted in. Amebiasis has a cyclic pattern and if stool examinations are made during a period when cysts or trophozoites are absent from the stool, results will be poor. Sawitz's experience is typical; he found that in normally passed stools, the percentage of positive diagnoses progressively increased from 21 per cent when one stool was examined to 76 per cent when six stools were examined. Moreover, the examination of a single stool following saline purgation was as effective as the examination of three normally passed stools. In this condition culture technics give only inconstant results and the complement fixation method is impractical for general use because of the difficulty of obtaining potent and stable antigen.

Shigellosis is diagnosed by cultural methods, the cultures being prepared by a technic which provides uniformly desirable colonies for examination. The best results are obtained with less inhibitory media, such as McConkey's agar, eosin-methylene blue and Endo's agar. The percentage of positive cultures is higher from an enema specimen than from the stool or from material secured by aspiration at sigmoidoscopy. Occasionally, the organism is identified on the first culture. In the writer's experience, an average of five has been required and in some cases as many as twenty examinations have been made before positive results were secured. Diagnosis is likely to be simplified if the examinations are carried out during an exacerbation of symptoms but in any case the secret of success is

repetition of the examination until the organism is isolated.

The diagnostic routine now employed for all patients who present themselves with a complaint of chronic diarrhea has evolved with experience, which has shown that the omission of any part of the study is likely to cause difficulties for patient and physician alike. The following investigations are carried out on successive visits:

1. Examination of a normally passed stool for protozoa and helminths; culture for Shigella of material secured from the rectal wall by the Hardy-Watt swab; tests for brucellosis.

2. Frei test for lymphopathia venereum; gastric analysis, to exclude achlorhydria; urinalysis; repetition of stool examination; repetition of culture for Shigella (Hardy-Watt swab).

3. Blood study, including mean corpuscular volume determination, to exclude sprue; repetition of stool examination; repetition of culture for Shigella (Hardy-Watt swab).

4. Sigmoidoscopy; examination of a stool passed after saline purgation for helminths and protozoa; culture of an enema specimen for Shigella.

The patient is prepared for sigmoidoscopy by a routine for which written instructions are supplied, as follows:

1. On retiring a dose of sodium phosphate sodium biphosphate (Fleet's phospho-soda) is taken. The cleansing effect is better than that of other saline purgatives and it seems to be ideal for encystation of Endanoeba histolytica; cysts are more practical for diagnosis than trophozoites. The medication is taken at night so that the patient is not aroused from sleep and so that the parasites during the night are washed from the lesions into the lumen of the bowel. If the purgative is taken in the morning, they have not had time for full maturation and are found as precysts, which do not lend themselves to differentiation.

2. A stool specimen is collected after 6 a.m. the following morning and is therefore fresh for examination.

3. Two enemas of physiologic salt solution in the amount of 1 quart each are taken in the morning; a 5 cc. specimen is collected from the final portion of the second enema. Culture of this specimen is particularly useful in the identification of Shigella.

4. Sigmoidoscopy is performed by the usual technic. Material is collected from demonstrable lesions if they are present and from random areas of mucosa if they are not. It is important that in amebiasis a special glass aspirator be used for the collection of the specimen; a cotton swab is not satisfactory because parasites are likely to be lost in the fibers.

A separate routine of testing is necessary for brucellosis. Cultural methods have been discontinued as not worth the trouble in chronic brucellosis although they may be valuable in the acute disease. It has been repeatedly shown, moreover, that the incidence of positive results is very low in abortus infections. The routine includes:

1. Agglutination tests, carried out by the Huddleston rapid slide macroscopic method with Lederle's antigen, which is quite as reliable and specific as slower technics: Different observers attach different degrees of importance to agglutination titers of various levels. Most observers regard titers of 1:80 as evidence of active Brucella infection while granting that they may occur in the presence of other infections and, of course, after skin tests for brucellosis or vaccine therapy with Brucella organisms. The writer formerly regarded all dilution titers of 1:160 or above as diagnostic of active brucellosis and titers below this level as suspicious. With increasing experience with the sensitivity test, however, an occasional patient with titers of 1:640 and more has proved not to have brucellosis. Several patients with titers of this level had negative sensitivity tests for active brucellosis; they were found to have amebiasis and were relieved of all symptoms with adequate treatment for that disease.

2. The opsonocytophagic index, which is based upon the ability of neutrophilic leukocytes to phagocytize Brucella organisms and which is an index of the degree of the patient's immunity rather than a true diagnostic test. Special laboratory facilities are required.

3. The intradermal test, by means of an intradermal injection of Castaneda's Melitensis, bovine, porcine vaccine. A positive test, which is presumed to indicate that the patient has, or has had brucellosis, depends upon the development of an erythema measuring more than 10 mm. at the end of twenty-four to forty-eight hours. The reaction is not regarded as negative until forty-eight hours have passed without the development of a clearcut erythema or of any erythema at all. A test which appears positive at the end of twenty-four hours but is negative at the end of forty-eight hours is interpreted as a negative reaction. In the writer's experience the intradermal test has proved a most satisfactory diagnostic method.

4. A sensitivity test, the principle of which is the injection at five-day-intervals of successively larger doses of the Castaneda vaccine until 2.0 cc. is reached. A single, clearcut reaction is regarded as diagnostic and therapy is substituted for further testing. If a clearcut reaction has not occurred before a dosage of 2.0 cc. is achieved, brucellosis can safely be eliminated as the cause of diarrhea. The reaction must be clearcut. A mild reaction is interpreted as negative for brucellosis.

Children are tested and treated with the Castaneda vaccine in the same dosages as are adults.

THERAPY

Initial Therapy. The first step in the management of a chronic diarrheal disease is, paradoxically, therapeutic, the administration of a multiple vitamin preparation of high potency. Diarrhea is a well known manifestation of such vitamin-deficiency diseases as sprue and pellagra and avitaminosis is a probable complication in a diar-

rhea of any origin which has lasted, to set an arbitrary limit for the establishment of chronicity, more than ten days. The hypothesis of a borderline avitaminosis is perfectly valid. When a person in this group develops diarrhea, it does not take long for the surplus of vitamins in the body, which is never very large, to be used up. The same process occurs in persons not in the borderline group whose diarrhea has been severe or has lasted over a considerable time. In neither type of patient can the vitamin needs of the body be met by the ordinary diet because, with a continuing intestinal disturbance, all food ingested passes rapidly through the intestinal tract and there can be no adequate absorption of vitamins. Even if the disease is self-limited, the deficiency continues to exist.

The patient with a continuous diarrhea, therefore, when he is first seen, is at once given a multiple-vitamin preparation of high potency. The results are frequently spectacular although they do not remove the responsibility from the physician for complete investigation. Incidentally, if the investigation proves negative and if there is no recurrence of the diarrhea, the results secured from therapy can justifiably be regarded as diagnostic.

Amebiasis. Every patient with amebiasis should be treated. This is not a disease which can be ignored with safety. The patient who harbors the infection is a source of infection for others. From his own standpoint he is either actually ill or is in a state of subclinical ill health from which serious complications may arise. It is open to question whether asymptomatic amebiasis very often exists. The patient classified in such a category frequently does not realize that he has had symptoms until he is relieved of them and enjoys the improvement in health and well being which follows.

In the order of effectiveness, the available amebicides are diodoquin, chiniofon, carbarsone and vioform although for some reason, which at the moment is not clear, diodoquin seems less effective than it formerly was. Emetine hydrochloride is of value in relieving the acute symptoms of either dysentery or diarrhea and it is the only drug effective in ameboma and in various extraintestinal amebiases. Its curative properties in intestinal amebiasis are probably less than 30 per cent.

Emetine, furthermore, is a protoplasmic poison and potentially toxic. Within a recent six-month-period, four patients were seen in consultation who, following its use, developed such severe symptoms that they had to be rehospitalized. The symptoms were numerous and bizarre and changed from day to day. They included cramps, headaches, dizziness and pains in various locations; they were associated with electrocardiographic changes and they were disturbingly slow in their disappearance. It may be that the dosage generally used at present (10 to 12 gr. in daily doses of 1 gr. each by the subcutaneous or intramuscular route; both are equally painful) is too high. As a matter of fact, very little is known about the optimum dosage; it is quite possible that effective results could be achieved with one-half or one-quarter of the dosage now being used.

If amebiasis is resistant to treatment and if after adequate investigation Shigella is not found in the occasional selected patient, combined treatment may be instituted (in the hospital), consisting of (1) emetine (1 gr. nightly for ten to twelve days) on the theory of eradicating the ameba via the blood stream; (2) penicillin (30,000 units intramuscularly every three hours for twelve days) to combat secondary infection via the blood stream and (3) neoprontosil (25 gr. four times daily for twelve days) to combat secondary infection via the intestinal tract. In addition, diodoquin is given three times daily in six-tablet doses while the patient is hospitalized, to eradicate ameba in the intestinal tract. The dosage is reduced by one-half after the twelve-day-period of hospitalization and is continued in that amount until the twentieth day. Neoprontosil is the best antidiarrheal drug presently available although its effect is probably on the associated pyogenic infec-

tion rather than on the amebic disease *per se.* Such combined treatment is extremely effective but it is also heroic and it should not be employed in the absence of special indications.

One point should be borne in mind in evaluating the therapy of amebiasis: Symptoms do not disappear until some time after parasites have disappeared from the stools. Post-treatment attacks, however, become successively milder until they eventually disappear altogether and therapy should not be reinstituted unless parasites again appear in the stools.

Shigellosis and Brucellosis. Sulfonamides and antibiotics are not effective in the chronic phases of shigellosis and brucellosis. Both diseases are treated with vaccines, administered subcutaneously in successively larger but cautiously increased doses at intervals of five to seven days, preferably the latter. The amount of the initial injection is gauged by the size of the skin reaction. Treatment of shigellosis is by an autogenous vaccine made up as soon as the organism is isolated. Treatment of brucellosis is by the M. B. P. vaccine (Melitensis, bovine, porcine) of Castaneda. In both diseases the patient is kept under careful observation, reactions to the previous injections are checked before succeeding injections, every patient is individualized and it is an invariable rule to employ a smaller dose whenever there is doubt as to the wisdom of a larger dose.

It is most important that these instructions be followed exactly. Dangerous reactions can ensue when they are disregarded and one fatality is known to have occurred under treatment in shigellosis which was charged up to the treatment but which should have been charged to the complete disregard of instructions.

An interesting observation during treatment in patients with brucellosis is the development of symptoms of which they had not previously complained, perhaps representing allergic reactions. If the explanation is valid, it supports the concept that the efficiency of Melitensis, bovine,

porcine vaccine rests upon its desensitizing rather than upon its immunizing action. Reactions are likely to be severe if all foci of infection are not eliminated before treatment.

Vaccine therapy in shigellosis and brucellosis is supplemented by the Jones sugar-free diet which is strictly adhered to until improvement in symptoms occurs. Then articles from a list of permitted foods are added one at a time and in large quantities so that if an idiosyncrasy to them still exists, it will be demonstrated at once. If sensitivity no longer exists, these articles can be eaten in the usual quantities.

It is imperative that patients treated by vaccine therapy understand that this is not a rapid method of treatment. In shigellosis, results are not apparent for six months and in the usual patient treatment must be continued for a year or more although success in children is occasionally spectacular in much shorter periods of time. Patients with brucellosis are not likely to see any improvement before the tenth week at least. A minimum of six months of treatment is necessary, even if results are observed earlier, and treatment for a year or more is not unusual. Patients who have both amebiasis and brucellosis will not, as already pointed out, be benefited by amebicidal therapy until brucellosis is adequately treated. Prompt and permanent disappearance of Endamoeba histolytica from the stools can then be anticipated after a single course of amebicidal therapy.

In conclusion, two rather uncommon types of diarrhea might be briefly mentioned which undoubtedly exist but which are difficult to establish etiologically: The first is the so-called geriatric type, which occurs in persons over fifty-five years of age and in which neoplastic disease is not a factor. A possible explanation is the assumption by the intestine of new functions. The course of events is essentially as follows: Nephrosclerosis of various degrees is a rather common finding in most persons fifty years of age and over. Its existence is

reflected in dye excretion tests, the results of which are likely to be lower than normal. The diminished renal function, however, is not, as usual, reflected in the non-protein nitrogen level, which is substantially normal. The explanation may be that the colonic mucosa, which has the ability to extrude nitrogenous waste products, compensates by the assumption of this function for the decrease in kidney function. The irritation which is caused by the presence of the abnormal nitrogenous waste products in the bowel is expressed clinically as diarrhea. The theory is additionally supported by the fact that diarrhea is frequently a part of the syndrome of uremia. Patients in this group almost invariably improve when their protein intake is reduced.

The allergic variety of diarrhea undoubtedly exists but it should be regarded as established only after all other diagnoses have been excluded by adequate testing. It must be distinguished from the dietary difficulties associated with colonic diseases of other etiologies. Patients with chronic diarrhea, especially when amebiasis is the cause, very frequently cannot eat certain types of food but their difficulties are apparently not due to allergy for when the basic disease is cured by the proper therapy—therapy which would not affect an allergic condition—they can eat foods quite comfortably which formerly disagreed with them. Even more convincing is a personal experience with Jones' sugar-free diet, which is used routinely in shigellosis and brucellosis. Very few patients with diarrheas of other origins have not been relieved symptomatically by this diet. Yet it contains both wheat and eggs, which are common offenders in allergy and which cause no upsets when they are ingested. Patients with the allergic type of diarrhea are usually not seen in consultation. Food allergy is seldom the first manifestation of the allergic condition and it is a relatively simple matter to correct it in the course of treatment of the underlying disease.

SUMMARY

The chronic diarrheas have been discussed from the standpoint of considerations which have presented themselves as important in a practice limited to diseases of the colon. Amebiasis, shigellosis and brucellosis are the most common causes of chronic diarrhea. All three present vague symptoms, much the same for each disease, and all three are capable of mimicking other diseases. Diagnosis is a laboratory matter and may be difficult. The therapy involved has been outlined briefly.

DISCUSSION

THOMAS T. MACKIE (Winston-Salem, N. C.): I have found Dr. D'Antoni's paper extremely provocative. I am indeed sorry that the nature of his paper has prevented inclusion of evidence which I would certainly like to see in support of certain of his points. I am very much in agreement with many of the things that he has said but I am in serious disagreement with a number of others. It is impossible in the time allotted for discussion to do more than to touch upon the points of disagreement. I want to emphasize that this is merely the expression of differences in experience and differences in interpretation.

In the first place, with respect to the definition or rather the etiology of diarrhea and dysentery, I take exception to the statement that the etiologic agents are different. For example, the Shigella group and likewise the Endamoeba histolytica may produce, each of them, diarrhea and dysentery. I agree very strongly with Dr. D'Antoni's statement that Endamoeba histolytica is invariably a tissue parasite and that consequently it should be treated whether the individual has intestinal symptoms or not. By far the great majority of individuals who are carrying this infection fall into the category, unfortunately, of the individual who is classed as a neurotic because his symptoms are vague, indefinite and not characteristic of any well recognized clinical syndrome.

The type of picture which Dr. D'Antoni has described in children is important because it is a reflection not only of the picture in children but in many adults and one which I have been seeing in the last few months in many of our overseas personnel who are returning, finding

themselves incapable of resuming the normal intensity of civilian life because of an unrecognized and unthought of amebic infection. All patients then with amebiasis should be treated.

Now, we come again to one of the points with which I am in serious disagreement, the matter of the effectiveness of different drugs and the toxicology of these drugs. Diodoquin is useful and so are the other members of the hydroxyquinoline sulfonic acid group. These drugs, however, are not absorbed from the intestinal tract in such amounts to reach the amebae which are actually residents within the host's tissues. If one visualizes the pathologic condition as one sees it under the microscope, the way these protozoates are not only on the surface of the mucous membrane but are in the mucosa and deep in the muscularis mucosa, in the tissues of the submucosa, no drug which is not absorbed and distributed by the blood and lymph streams will be effective against organisms in that site. That statement applies to diodoquin, vioform and chiniofon as well as to the old German yatren.

The second group of drugs, of which there is only one member that should be used today, is carbarsone, an organic arsenical. That perhaps most nearly approaches the ideal amebicide because it is to some extent effective in both anatomic areas, that is to say the surface of the mucosa and within the tissues of the colon itself. But it is not completely effective and if one follows adequately—and by adequately I mean over a sufficient period of time—individuals resident in an area where they are in theory at least not subject to reinfection, one finds a relatively high recurrence rate.

Emetine, in my judgment, is certainly the most useful drug that we have because it is the only drug which is fully effective against the organisms which are in the tissues. Any of you who have treated amebic abscess of the liver know that. Its toxicology is very well defined. It is a protoplasmic poison; consequently, (and this is a general rule of therapy) any drug which is known to be toxic to host tissues should not be given in terms of so many grains per day or for a week, it should be given in ratio to the patient's body weight. That is fundamental. It has been thoroughly established with this drug that if the total daily dosage does not exceed 1 mg. per Kg. of body weight and if the total dosage over any course does not exceed 10 mg. per Kg. of body weight,

one will not encounter toxic effects in the host that are a cause of any anxiety whatever.

The dosage that Dr. D'Antoni speaks of, 12 gr. in one course, in my judgment, is definitely higher than it should be. In his manuscript in the discussion of the complicating factors, and particularly the coincidental infection with the Shigella, the organisms of bacillary dysentery, he advocates the use of neoprontosil but in his paper of the morning he advises the use of vaccine. It has been the experience of the armed forces that the whole group of Shigella responds extraordinarily well to certain of the sulfonamide drugs. Perhaps the two or three most effective are sulfaguanidine, sulfadiazine and sulfathiazole. It seems paradoxical to me that he should recommend one of these drugs for infections complicating amebiasis and yet in his pure Shigella diarrheas advocate the use of autogenous vaccine.

Furthermore in my own experience, in long extended clinical trial of these cases using autogenous vaccines, I have found them not infrequently dangerous, as Dr. D. Antoni pointed out, but I likewise have been very unimpressed as regards their efficacy in eliminating the infection on the one hand or really relieving the patient's symptoms on the other.

This matter of brucellosis, as I have previously said, is one of the most provocative things in this paper. It is rather a curious situation it seems to me that with the importance which this disease has assumed in the United States since 1920 and the number of cases which have been reported that the syndrome of a chronic diarrhea due to that organism has not been recorded previously. That, of course, brings up the matter of diagnosis.

Dr. D'Antoni, I frankly think, rather unwisely has eliminated in his manuscript the one method by which a positive diagnosis can be made, in other words, the effort to recover the etiologic organism. The methods on which he depends are slide agglutination. The slide agglutination of itself is notoriously inaccurate. He depends upon the intracutaneous or cutaneous test using the killed organisms. That, if it is positive, merely proves an association and that association may not have been in its most active phase associated with a clinical response. It merely indicates that there has been a biologic relationship between a host and that particular organism.

He also includes the application of the

opsonocytophagic index, a procedure of considerable difficulty to carry out, the interpretation of which is still not without a certain amount of doubt and hazard.

The net result of it is that, in my judgment, there are only two methods by which a diagnosis of brucellosis can be made. The first, naturally, is the recovery of the organism; the second—and this is reasonably accurate—if one can show in the particular patient using the standard tube agglutination test that there is a rising titer of agglutins over a period of two or three weeks, then one may assume that one is dealing with an active and positive infection.

His treatment, again by vaccine, is disturbing. There are many treatments for brucellosis: Foshay's immune goat serum, all of the antibacterial drugs and antibiotics, the injection of cultured filtrates and, of course, the continued injection of autogenous vaccine over long periods of time. Again, there is a good deal of conflict in the experience of others as to the real efficacy of that type of treatment.

I want to say just a word about the giardiatie diarrhea. I would like to know if there is any evidence for it. I personally have never seen a situation in retrospect that I could possibly explain on such a hypothesis. I question the wisdom of submitting such sweeping hypotheses with no evidence in support.

May I conclude then, by complimenting Dr. D'Antoni on a very interesting paper and repeat again that what I have said is not meant in any sense as unkind criticism; it is simply a frank expression of my own interpretations of these problems.

Mary M. Spears (Philadelphia, Pa.): I am very much interested in Dr. D'Antoni's paper on chronic diarrheas, particularly as it relates to their etiology.

The incidence of the types described by the author is small in Philadelphia compared to that in New Orleans. The patients I see with chronic diarrhea fall into a miscellaneous group etiologically, with a low percentage due to infection. I should like to confine my remarks to the cases with which I am familiar.

I agree with Dr. D'Antoni that chronic diarrhea is not a clinical entity but is an expression of an underlying cause which is not necessarily in the colon. Since the etiology is difficult to determine, the patient should be considered as a whole and not from the condition of the colon alone. Also emphasis should

not be placed entirely on the laboratory for diagnosis.

Obviously a careful history of symptoms, including eating habits, personal hygiene, type of employment and a thorough physical examination are important. While the social status is important, I have found that diarrheas do not occur exclusively among those who eat too little and therefore suffer with malnutrition, but perhaps more frequently among those who eat too well. A diary of food and symptoms is often very illuminating and will throw convincing light on the cause of many cases.

Allergy as a causative factor in diarrhea cannot be dismissed. From my observations allergy is quite near the top of the list in the etiology of this condition. Some of the offending foods that have been exposed are wheat, citrous fruits, dairy products, sugar, carrots and some drugs. In this connection I think it is important to know what medication the patient has been taking. We have found the diarrhea in a given case to be cured by the withdrawal of sedatives and vitamins to which the patient was allergic.

We should know something about the emotional background, incompatibilities and conflicts in the homes and the places of employment of our patients. In the patients in whom the emotional basis is the factor the condition is secondary to the anxiety state and not the anxiety state secondary to infection. It has been demonstrated many times that when the environment has been changed and the situation relieved the diarrhea has promptly cleared up.

A physical examination has revealed an undiagnosed hyperthyroidism which was the cause of the diarrhea for which the patient sought relief; a cure took place when the underlying cause was treated. The cholecystectomized patient is often the victim of a diarrhea which is resistant to treatment.

A gastrointestinal investigation should include biliary drainage, gastric analysis, feces examination and rectosigmoidoscopy. The first will sometimes expose giardia which in some of the cases we have seen have been the etiology; achylia has been the cause in some patients and carbohydrate fermentation the basis in a large percentage. We are all familiar with the tragedies of patients who have been treated and not examined and in whom an inoperable carcinoma is found. In our patients

who have all had complete studies the number due to bacterial infection or parasitic infection is very small. X-ray examinations were done as indicated.

I cannot recall a patient with diarrhea due to avitaminosis, but I have treated patients with it caused by a vitamin. I think we should be careful and alert in our medication.

I appreciate the privilege of participating in the discussion of this interesting paper and this important medical problem.

MARTIN S. KLECKNER (Allentown, Pa.): It has afforded me great pleasure to hear Dr. Antoni's paper on chronic diarrheas. As a man who has had tremendous experience in the field of parasitology and pathology, he has brought to us a review of some of the important phases produced by this symptom-complex. I do not agree in full entirely with everything that Drs. D'Antoni or Mackie have said so that I will pursue a middle of the road attitude. I have found this very interesting, and at times, distressing symptom to exist in 15 per cent of my patients in protologic practice.

Due to airplane travel and the return of many of the servicemen following peace in the last world war, many of the tropical diseases which had not been as prevalent heretofore have either been greatly increased or their presence has been brought more strongly to our attention.

I wish to enumerate several points concerning which you as proctologists treating diseases of the anus, rectum and colon must always be cognizant: Careful history taking is very important as well as complete physical and proctologic examination which includes sigmoidoscopy. We are dependent in great measure on the pathologic and roentgenologic laboratories. Those of us who are fortunate enough to have trained personnel in our laboratories that understand parasitology and those who believe in the diagnostic value of the double contrast barium air enema are indeed most fortunate.

Examination in the pathologic laboratory of warm stools from clean containers produces the best results if analyzed within the first forty-five minutes. I do not believe the taking of smears for culture and scrapings via the sigmoidoscope has been mentioned.

Diarrhea is due to a number of causes. Some of them may border on the acute but we must bear them in mind. They are as follows:

1. Ingestion of improper food and drink. Food poisoning and prolonged overindulgence of poor grade liquors should be mentioned.

2. Deficiency diets when avitaminosis plays an important part. Pellagra is a good example.

3. Diarrheas due to invasion and infections by certain organisms of bacterial and protozoal origin. My experience is very limited regarding shigellosis and brucellosis but the amoebic and other forms of bacillary dysentery have been seen. These must be remembered in diagnosing the diarrheas. Do not forget that the "carriers" of these conditions may need more personal care than many others. Certainly our health examinations in regard to handlers of food and drink should receive more attention than ever. Stool cultures and smears are often disregarded in this modern era when health and longevity are emphasized. Under this category must be mentioned chronic ulcerative colitis many times characterized by eight to fifteen daily bowel movements.

4. Giardiasis and the malarias are mentioned ·because they may produce severe chronic diarrheas.

5. Anatomic derangements or lesions in the colon such as diverticulitis, the presence of polyps and malignancy may all cause chronic diarrhea. One thing we often forget is the presence of the most common foreign body in the rectum, fecal impaction. Some of these very large impactions in patients may produce a diarrhea, or a tenesmus simulating diarrhea for weeks and may require the injection of peroxide of hydrogen, oil enemas, etc., to break it up. Sometimes a patient must be put under an anesthetic in order to evacuate the impacted feces.

6. There are various nervous influences that may produce a diarrhea and we are prone to call it (erroneously) mucous or functional colitis.

7. Long administration or overdosage of certain medications such as arsenic and mercury and the much abused mineral oil. The active use of radium and deep x-ray therapy may cause what is known as a factitial proctitis with diarrhea as a common symptom.

8. Parasitic infestation listing the helminths, the cestodes and the nematodes as primary causes is common but so little understood. I regret that time does not permit further elaboration of this condition.

Let us insist upon complete investigative methods that all chronic diarrheas may be more carefully diagnosed and treated.

Joseph S. D'Antoni (closing): I think it is most unfortunate that I have been negligent over the years in publishing a lot of the work that we have been trying to do and tried to get to the bottom of. Consequently, I think that some of Dr. Mackie's statements could be blamed on this fact; namely, that it is introduced in very short sentences and very little or no background for our having come to those decisions has been made in the paper. I am not blaming him for not reading the literature. I am blaming myself for not having published some of this work and given more definite proof, as he asked for.

First of all, regarding diarrhea and dysentery and my statement saying that it should be differentiated because the etiologies are different, I am speaking at that point of the group of etiologies. When you have dysentery, your patient is automatically transferred into a particular group and when you have chronic diarrhea, he is thrown into another group; when you have acute diarrhea, he is thrown into a third group. I did not say that the etiologies in all of these groups were different. I have taught for years and believe I am correct in the statement that in the southern United States, 90 per cent of all dysenteries in the acute and chronic form are due either to Leviek or Shigella. In the northern United States, that statement would have to include your non-specific ulcerative colitis which, as I have said, we do not see. In certain parts of the area where flukes are present, namely, the Schistcsomum mansoni infections and japonicum infections, then if you have dysentery, you must include it as one of the etiologic agents. But it has always surprised me intensely to have patients with dysentery referred and a diagnosis made of some etiology that cannot produce dysentery. If the man who referred that patient had known that many of these etiologies do differ, that mistake would never have been made.

As to the effectiveness of drugs, of course, I have been one of the big boosters in this country of diodoquin. The British have used it recently. So in contradiction to Dr. Mackie's statement that iodine preparations will not cure, for the first time in the history of our knowledge of amebiasis the British are curing 75 per cent of their amebic patients who live in India, renowned for the most resistant strains in the world. If they add penicillin to it—and penicillin, as I have said in the paper, just as neoprotosil, is used to combat secondary infection—that cure rate has been made much higher.

I frankly doubt and I have one very capable backer in the person of Colonel Craig who has made the very definite statement that he doubts that 20 per cent of the patients with amebiasis are cured with emetine. I more or less have to agree with him on that basis aside from its toxic effect. I see Dr. Sodeman in the audience who can vouch for one lady who on the second grain of emetine (a woman of about 128 pounds) developed paroxysmal tachycardia and electrocardiographic changes. We have seen a number of patients of that type and we expect to see more if we are going to continue using this drug.

I made the statement very definitely in my paper that I believed neoprontosil was the best antidiarrheal drug, meaning that it was given to combat a symptom and not as a curative agent.

Secondly, we have always used autogenous vaccine and have always believed it to be the most efficacious.

On the question of brucellosis, Dr. Mackie, we have—and I reported at the Congress on Brucellosis in Mexico last November—twenty-six patients with intestinal brucellosis who had been observed for a period of six months to three and one-half years under vaccine treatment. These patients, I believe, have gone sufficiently long to say most probably we are correct. We have to wait ten or fifteen years probably to say that unequivocally but on the basis of what I have seen of diarrheas, on the basis of what you can learn in the literature regarding brucellosis, if you read about the pathologic conditions you will find that every organ in the body has been found to harbor this bug. I would like to be able to say that we can culture 90 per cent of the organisms present in all those with brucellosis. On the other hand, I doubt very seriously if we can culture the causative organism in more than 2 or 3 per cent.

I think in this country that 10 per cent of the chronic abortus infections can be cultured from the blood stream and that is by some very capable workers such as Huddleson, Forshay, Harris, Alice Evans and so forth. If we are going to rely on the culturing of brucella abor-

tus from the blood stream, then we are going to have more neurogenic diarrheas, as I have already stated, and stated why I thought they occurred so frequently.

Now, the vaccine that Castaneda has proposed is an endotoxin. I did not discuss it in the paper because it is in the American literature, primarily in the American Journal of Tropical Medicine.

As to the giardiatric diarrhea, I could defend myself by telling you a personal experience that occurred between my junior and senior year as a student. I worked in the pathology department at Charity Hospital and during the three months' period we had, I believe, 225 postmortems that were usually done within thirty to forty-five minutes after the patients had died. In those days the independent service in the hospital did all the postmortem examinations and we made it a rule to get through with these examinations as soon as possible so as to keep in the good graces of the under-takers. In seeing the bowel of uremic patients and in seeing the bowel of people over fifty years of age who had diarrhea, we frequently saw a yellowish, brownish-yellow discoloration in the submucosa and no textbook on pathology would tell us the pathologic condition that was in process. For some very peculiar reason which I am at a loss to explain, and I have asked many pathologists for an explanation, this brownish, peculiar submucosal discoloration disappears about one hour to an hour and one-half after the patient is dead, so that you must obtain the bodies "hot," in other words, in order to see this.

The fact that the colonic mucosa is capable of extruding nitrogenous waste products I think is an accepted fact among pathologists. I have limited it here to the colon. However, most pathologists believe and have shown, and it is a common fact among them, that non-protein nitrogen is extruded through the mucosa of the gastrointestinal tract.

There is one thing I would like to say. In the paper I have tried to include as many things as possible simply to develop in the physician an interest in the chronic diarrheas instead of this procedure of throwing his hands up and saying, "Neurosis!" My secretary will tell you that I have the craziest people or more crazy people than any three psychiatrists in New Orleans, but it is remarkable how, after you rid them of

their colon disease, mentally well these individuals become.

My remarks are not limited to patients from New Orleans. I have had the fortunate experience of seeing many patients from the tropics and from all over the United States. In Philadelphia, as in every other part of the United States, amebiasis is present. If you have a diarrhea—and I do not care what part of the United States or the world it is—the first thing you think of is amebiasis. I think if you continue looking for it and can get your doctors and the pathologists interested in it, you will find a much higher incidence than is presently reported.

I made the statement and thought I had made myself clear regarding the allergists. My experience has been this, that most patients are referred by other physicians and since they are referred and since so many of them have been to three, four, five, eight and ten doctors, the allergic diarrheas have already been picked up before we ever see them. I think there is no question but that it does very frequently occur. In my experience, and for the reasons which I have just enumerated, I do not see them very often.

As to the question of physical examination in these patients, I am not proud of the fact but because of the great tediousness of this work I have been unable to do complete physical examinations on these individuals, but I have always made it a practice to see if the physician recommending or referring the patient has not put that patient through the mill; most of them, incidentally, have been in hospitals five or six times; if the physician referring the patient has not had that patient in the hospital and given him a good work-up, he is always referred to a capable internist. We never begin looking for the cause of diarrhea until that internist will tell us, "Well, I think it is colon infection," not coming from such systemic disease as endocrine glands and so on and so forth.

Several years ago a urologist sent me a patient who had arthritis of the right hip. In subjects with shigellosis, we have seen a few patients with colon infection that had marked hypertrophic arthritis and because of the results that we obtained with this patient, one in particular and two others that were from this one urologist, he became very conscious of

the fact that if patients have colon disease, they may have a shigellosis and in turn that may be what is aggravating their arthritic manifestation. He sent me such a patient. The patient had not had a physical examination in several years. Therefore, I sent this patient to an internist. The internist examined the hip and found that there was marked limitation of motion. She had been having this trouble for about three months. He ordered an x-ray first and found that the femur was fractured and that was the arthritis that was being treated.

Consequently, I have a firm belief that all patients should be seen by an internist and the most capable one that you can find, if you do not do them yourself.

As for the giardial diarrheas, several years ago we were quite interested in this and checked back on our figures. In 5,000 patients with giardiasis that we had seen, over approximately a nine-year-period, there had been only three that had diarrhea from Giardia. Consequently, we always wait until we have ruled out every other etiology and if we can find no other etiology, then we say it may be the Giardia and treat the patient accordingly.

As to Dr. Kleckner, I would like to bring out one point which I did not mention in my paper. That is the complaint of the returning serviceman. His complaint is usually severe abdominal cramps and a tendency toward constipation rather than diarrhea. I would guess that in the probable seventy-five or one hundred returning

servicemen I have seen, about 75 or 80 per cent of them have had this one complaint of abdominal pain which is not at all infrequent but it is certainly not that common in all patients with amebiasis.

Regarding some of the topics or some of the diseases that Dr. Kleckner mentioned, we have made it a habit to place those in the acute diarrheas such as food poisoning, the poisoning of cadmium and other chemicals, such as arsenic and fluoride, and so forth. They, I believe, come under the heading of the acute diarrheas rather than the chronic diarrheas.

Likewise, I believe that fecal impaction, the patients I have usually seen, rarely run for a month. Consequently, they, again, are transferred into the group of acute diarrheas.

Finally, I would like to comment on the helminth infections: Needless to say, schistosomiasis is notoriously a producer of diarrhea and dysentery. The Strongyloides infections, which are fairly common in this country, also notoriously produce diarrhea but of the helminths, we can say this in general, any helminth is capable of producing diarrhea. Consequently, if you can find no other cause, regardless of how light your infection is, if you find, for example, only one or two eggs of hookworm on a zinc sulfate centrifugal flotation specimen, treat the patient for those hookworms, because that may be producing diarrhea.

In summary, let me again thank the three doctors who discussed this paper.
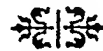
EVALUATION OF THE ROENTGENOLOGIC DIAGNOSIS OF LESIONS OF THE RECTUM AND SIGMOID

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LIKE many familiar phrases, that which defines the specialist as one who grows to know more and more about less and less is not as trite as it sounds and certainly contains some elements of truth and common sense. Those of us in the more limited specialties must be constantly alert to maintain a sound sense of balance.

The purpose of this report is to emphasize the need for co-operation between the roentgenologist and proctologist in that small group of cases in which all available information is necessary in arriving at an accurate diagnosis. We can only deplore the prevailing tendency on the part of the patient as well as his attending physician to accept the negative x-ray findings as infallible.

It is not within the scope of this paper to dwell in detail upon any of the technical phases of x-ray examination of the colon. However, emphasis should be placed upon the diagnostic superiority of the air-contrast technic over the routine barium enema, particularly when the latter has failed to show any pathologic condition in the presence of definite symptoms. The air-contrast examination requires additional time and effort on the part of the roentgenologist and is often an extremely uncomfortable experience for the patient. Nevertheless, the judicious employment of this technic will provide an accurate diagnosis resulting from the persistence, patience and skill of the conscientious roentgenologist. The latter is always ready to point out to the family physician the diagnostic inadequacies of the x-ray in the rectum and rectosigmoidal area and frequently suggests further examination by direct vision through the sigmoidoscope.

In order to illustrate the advantages of close co-operation between the roentgenologist and proctologist, representative cases have been selected from that group in which every possible diagnostic aid has been necessary to attain the final diagnosis. Although the space restriction permits the use of only one plate for each case, several films were taken of every patient chosen for presentation here.

Congenital Malformations. The x-ray can frequently furnish valuable preoperative information concerning various congenital malformations of the anus and rectum. When called to see a newborn baby with an imperforate anus and no demonstrable outlet, the surgeon is confronted with a surgical emergency that calls for all the diagnostic aids at his command. It is particularly in a deformity of this type that x-ray is useful in demonstrating the location of the blind sac. Knowing this, it can readily be determined whether the sac can be reached through a perineal incision or whether it is so high that such an approach would be useless.

The maneuver suggested by Wangensteen and Rice[1] for determining the distance of the blind sac from the perineum in a newborn infant with an imperforate anus and no demonstrable outlet is a very useful procedure. The infant is held by his feet long enough to allow any gas that might be present to rise and accumulate at the blind end. The relation of the air bubble to an opaque object fastened to the perineum determines the level of the blind sac. The writer has found this technic useful on three occasions.

CASE REPORTS

CASE I. In this first case a congenital atresia of a segment of the colon was suspected.

The infant was seen about eight hours after delivery and during that period no meconium had been passed and the abdomen had become distended. A normal anal outlet was present and a catheter could be inserted for a short distance. A plate (Fig. 1) was taken to see if any relationship could be shown between the level of the catheter tip and an air bubble at the obstructed area. This could not be demonstrated and the film served only to confirm the impression that any obstruction present was well within the abdominal cavity.

CASE II. A newborn infant had an imperforate anus with a perineal sinus so small that only a small probe could be inserted. (Fig. 2.) A dilute solution of a radiopaque oil was injected into the opening to demonstrate the low lying sac shown here. Knowing the position of the blind gut, it was possible to reconstruct an anus at the normal site at once rather than to temporize by enlarging the perineal opening.

CASE III. A nine year old girl was seen for the first time with symptoms of a low grade obstruction. Her only terminal outlet was a perineal sinus too small to admit an examining finger or an instrument. After the obstruction had been relieved by medical measures the x-ray demonstrated the tremendously dilated left colon pictured in Figure 3. It was evident that the dilated large bowel was caused by chronic obstruction at the outlet and was ap-

FIG. 1. Case 1. No air bubble could be demonstrated to show the site of the obstruction but the position of the catheter confirmed the impression that the point of obstruction was well within the abdominal cavity.

FIG. 2. Case II. Opaque media injected into the perineal sinus of a newborn with an imperforate anus revealed a low sac that could be approached through the perineum.

FIG. 3. Case III. The dilated left colon in a nine year old girl was readily demonstrated by x-ray although the only bowel opening was a perineal sinus too small to admit an examining instrument.

parently not a true case of congenital megacolon.

Lesions beyond Reach of Sigmoidoscope. The value of the roentgenographic findings in those patients where the lesion lies beyond the reach of the sigmoidoscope is obvious. However, two cases are presented to illustrate the dependence upon the x-ray in accurately diagnosing lesions above the lower sigmoid.

CASE IV. The plate shown in Figure 4 was taken as a routine procedure following the sigmoidoscopic discovery of the pedunculated adenoma that is demonstrated on the film just above the rectosigmoid. The presence of a second polyp beyond the reach of the instrument demonstrated the necessity for an abdominal approach although the lower lesion could have been removed through the operating sigmoidoscope. An interesting side light in this case was the fact that the roentgenologist, by maneuvering the second polyp under the fluoroscope, was able to estimate the length of the pedicle to within a centimeter of its actual size as later found at operation.
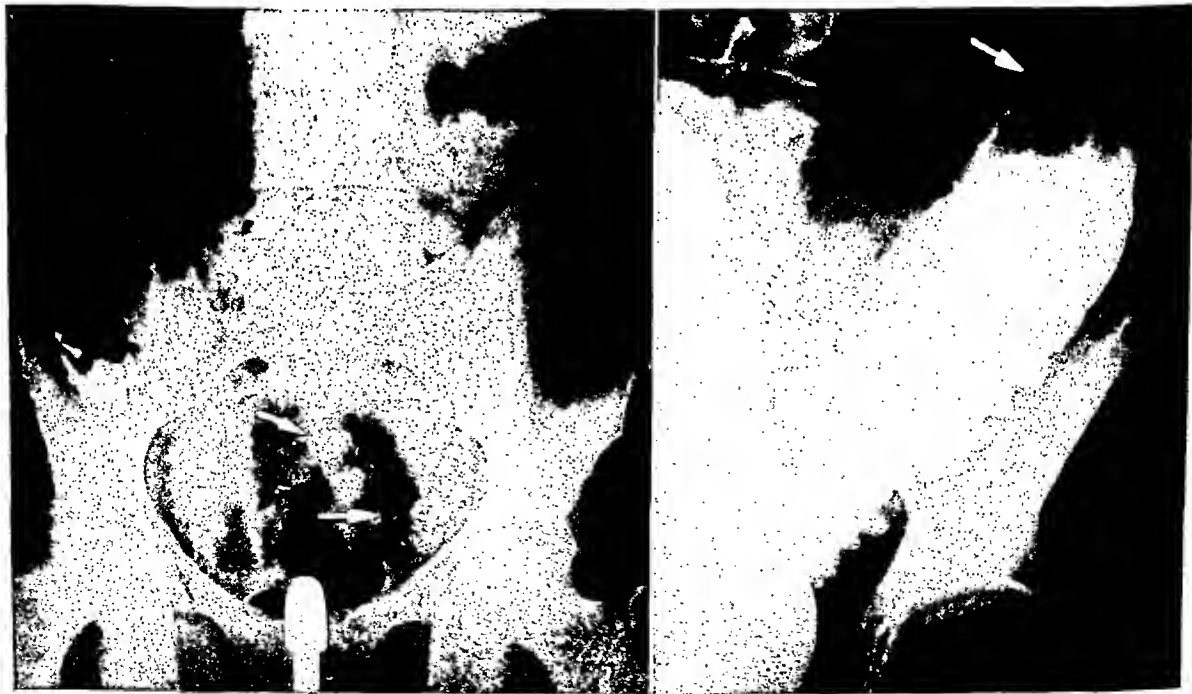
FIG. 4. Case IV. The arrow points to two adenomas of the sigmoid, only one of which could be reached through the sigmoidoscope.

FIG. 5. Case V. This film gives x-ray confirmation of a tumor of the descending colon tentatively diagnosed on sigmoidoscopic examination by the presence of blood-tinged mucus in the lower sigmoid.
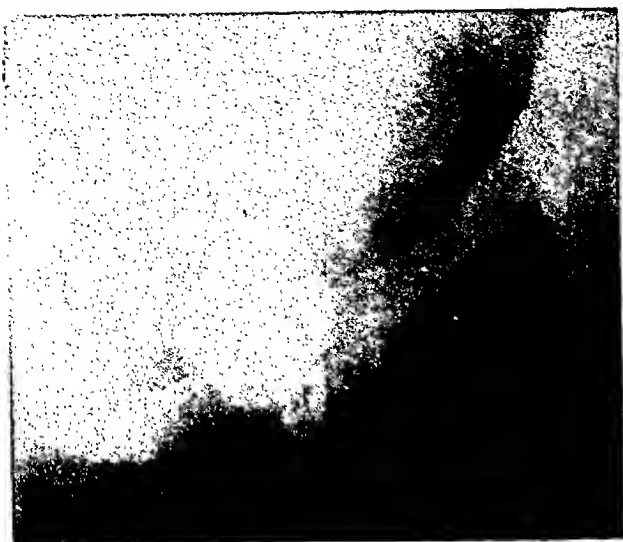
FIG. 6. Case VI. Roentgenographic evidence of a chronic supralevator cavity complicating an old ischiorectal abscess. The cavity above the levator had not been detected at two previous operations to obliterate the ischiorectal sinus.

FIG. 7. Case VII. Although this nine year old boy had had three unsuccessful previous operations for a fistula-in-ano, the x-ray showed that the persistent tract was a fairly simple one.

FIG. 8. Case VIII. The presence of the opaque material anterior to the coccyx proved the existence of an unhealed cavity although the patient had been discharged as healed the day previously.

CASE V. In this case the x-ray confirmed the tentative diagnosis of a tumor of the descending colon and demonstrated its approximate location prior to operation. (Fig. 5.) On earlier sigmoidoscope examination, only blood-tinged mucous was found to suggest the presence of a lesion beyond reach of the instrument.

Fistulas. Although the ordinary diagnostic measures are usually adequate to provide all the information necessary in the management of anorectal fistulas, the surgeon is occasionally confronted with a patient presenting an unusual problem. Three such cases have been selected to illustrate the additional information to be gained from an x-ray examination of the tract following the injection of a radiopaque oil.

CASE VI. About six months before this film was made (Fig. 6) a large ischiorectal abscess had been drained in the usual manner. A subse-

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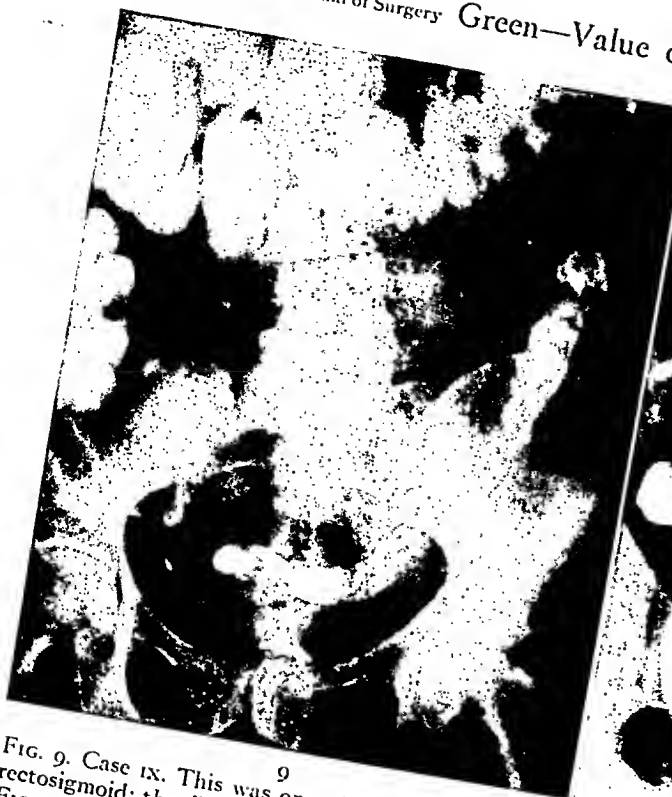
FIG. 9. Case IX. This was one of several films which failed to show the presence of a carcinoma just above the rectosigmoid; the diagnosis was made on sigmoidoscopic examination.
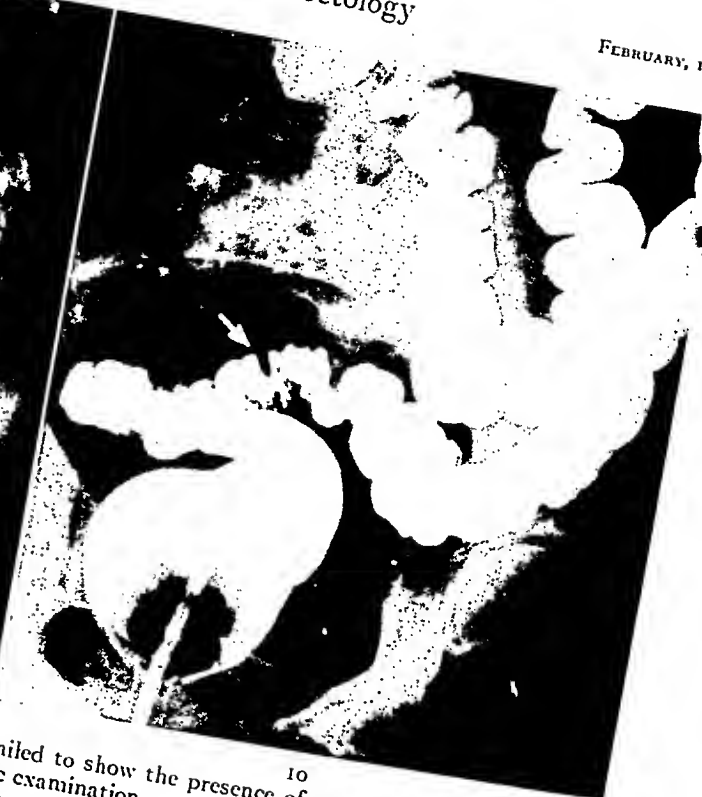
FIG. 10. Case X. The lesion at which the arrow points was diagnosed by x-ray and fluoroscopic examination as a carcinoma of the sigmoid. The sigmoidoscopic appearance, augmented by biopsy, proved that the defect was caused by fibrosis secondary to a chronic diverticulitis.
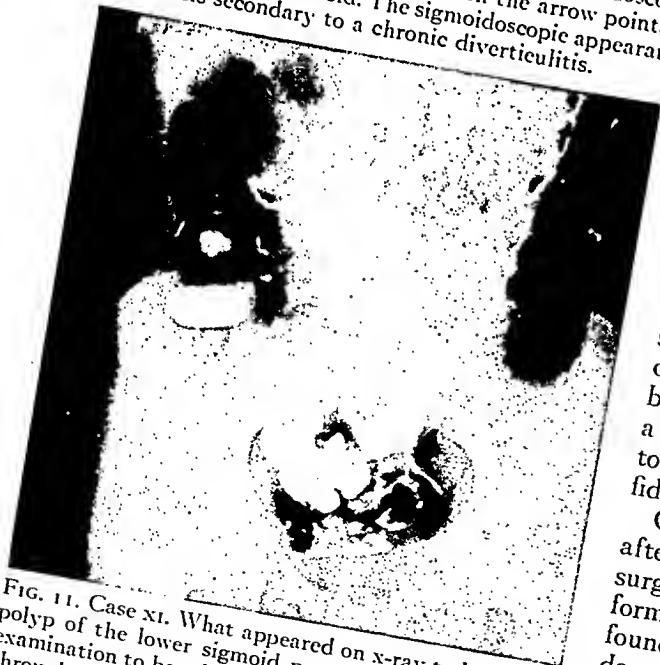
FIG. 11. Case XI. What appeared on x-ray to be a large polyp of the lower sigmoid proved on sigmoidoscopic examination to be a knuckle of sigmoid intussuscepting through the rectosigmoid into the rectal ampulla.

quent fistulectomy with obliteration of the internal opening was carried out about five weeks later. Healing progressed to a point where a small sinus persisted in the scar over

the ischiorectal fossa. Probing afforded no information as to the reason for incomplete healing. However, the injection of an opaque oil demonstrated a large supralevator cavity which had not been discovered at the two previous operations.

CASE VII. Prior to the first examination, a seven year old boy had had three unsuccessful operations for a fistula which was first noticed before he was a year old. Figure 7 demonstrates a fairly simple tract which enabled the surgeon to begin the operation with a feeling of confidence he might not have otherwise had.

CASE VIII. The patient was seen the day after he had been discharged as healed by a surgeon who had six weeks previously performed a fistulectomy. A defect in the scar was found and the injection of an opaque oil demonstrated the cavity shown in Figure 8. Because of the possibility that the case might develop an important legal aspect, the x-ray was valuable as additional evidence to substantiate the diagnosis.

Incorrect X-ray Diagnosis. Up to this point the cases presented have illustrated conditions in which the proctologic diag-
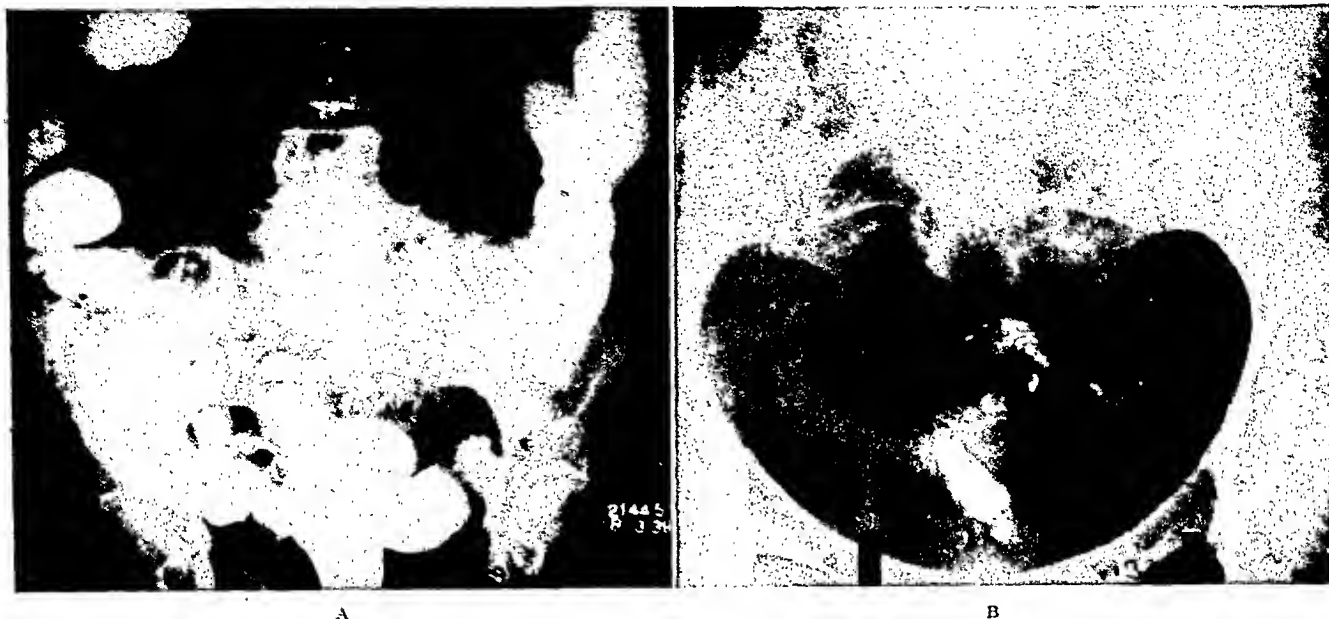
FIG. 12. Case XII. A, this film of a routine barium enema failed to show any disease in the sigmoid area; B, an air contrast study on the same patient revealing one large polyp in the sigmoid.

nosis has been supplemented and aided by the x-ray findings. The following group is composed of instances in which the x-ray diagnosis was inaccurate and the correct diagnosis was reached only after a sigmoidoscopic examination had been made.

CASE IX. This case is presented to emphasize the inadequacy of the x-ray in accurately diagnosing lesions in the area just above the rectosigmoid. In this instance no lesion was discovered on x-ray but the symptoms were such that proctologic consultation was requested *at the insistence of the roentgenologist.* Sigmoidoscopic examination demonstrated a carcinoma in the area above the rectosigmoid that was difficult to visualize on x-ray examination. (Fig. 9.)

CASE X. A roentgenologic diagnosis of carcinoma of the lower sigmoid was made after careful and repeated x-ray examinations. However, when a sigmoidoscope of small caliber was passed into the lumen of the stenosed area, pus was seen escaping into the bowel from a small diverticula opening; a biopsy specimen was also taken from the wall. The defect, diagnosed as a carcinoma on x-ray examination (Fig. 10) was shown to be caused by the fibrous thickening of the wall secondary to a chronic diverticulitis.

CASE XI. This case was a beautiful air-contrast demonstration of a polyp prolapsing into the rectal ampulla through the rectosigmoid. The child was first seen by a pediatrician who
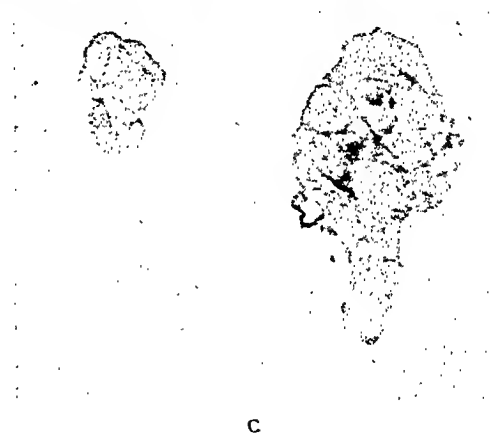
FIG. 12. Case XII. C, the larger polyp pictured in this photograph is the one shown in the air contrast film. (Fig. 12B.) The second and smaller polyp was not demonstrated by x-ray but was found at operation just 2 cm. above the larger one.

replaced what he thought was a polyp that was protruding through the anal orifice. The x-ray film (Fig. 11) confirmed the diagnosis and it was decided that such a low-lying tumor could best be removed through the operating sigmoidoscope. The age of the child was such that the passage of the scope without anesthesia was inadvisable. Preparations were made to remove the tumor under anesthesia and the scope was passed to a distance of 20 cm. from the anal margin, a point well above the site of the polyp as shown on x-ray examination. However, no tumor was seen although its presence was again confirmed by another x-ray. At this point, a general surgeon who was called in consultation felt that the roentgenographic find-

ings justified an exploratory laporotomy. At the insistence of the child's father who was a physician, another attempt was made to locate this apparently low-lying polyp. When it was not found, the anesthetist was asked to lighten the anesthetic so that the child would strain and push down any tumor that might lie above the reach of the scope. No polyp was seen but as the instrument was withdrawn a knuckle of sigmoid intussuscepted through the rectosigmoid to simulate the "polyp" so clearly demonstrated in Figure 11.

CASE XII. The three photographs in Figure 12 belong in a series that again emphasizes the fallacy of complete acceptance of the roent-genologic findings. Figure 12A is that of a routine barium enema ordered because of occasional, slight bleeding with defecation; nothing was found to explain the symptoms. After the examination was repeated using the contrast technic the presence of the large tumor seen in the lower sigmoid was clearly demonstrated in Figure 12B. The larger of the two polyps shown in the photograph (Fig. 12C) was the one seen in Figure 12B after it had been resected through the sigmoidoscope. Following its removal, the smaller one was discovered less than 2 cm. above it. It was apparent that the larger tumor had completely obscured the smaller one.

Psychic Factors. This presentation would not be complete without mentioning that large group of patients in whom an x-ray examination of the colon is necessary to convice them that no disease exists. Roentgenographic findings in this group are usually negative. In these times when the subject of cancer is so well publicized all physicians are familiar with the apprehensive individual, usually afflicted with a spastic colon, who will accept the fact that no tumor exists only after he has undergone a colon x-ray. Some individuals who have had an abdominoperineal resection for carcinoma of the terminal bowel are sometimes obsessed with the idea that they have developed another cancer. On a few occasions it has been necessary to do a colonic filling through the colostomy stoma in order to convince these unfortunate patients that no other lesion exists. A thorough physical examination, including the passage of the sigmoidoscope, fails to impress them as does the huge machine that crackles and sputters in the darkness of the fluoroscopic room.

CONCLUSION

This has not been the type of report from which one can draw any well defined conclusions. Each physician must determine for himself from the history and physical findings in the individual case whether or not an x-ray examination will be of benefit to his patient. It is a waste of time and money to order colonic fillings routinely just as it is criminal neglect to omit that examination if it can be useful in arriving at an accurate diagnosis. By working in close cooperation in the type of cases mentioned here, the proctologist and roentgenologist can arrive at a more accurate diagnosis than either could have reached alone.

Credit is given to Drs. M. E. Goodrich, W. S. Peck, C. E. Hufford and F. C. Curtzweiler, of Toledo, for the use of their films for reproduction. Close cooperation with these roentgenologists has been stimulating to the author and has resulted in a more accurate diagnosis in the type of cases presented.

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DISCUSSION

JEROME WAGNER (New York, N. Y.): The privilege of commenting upon the presentation of Dr. Green is an opportunity to add emphasis to our obligations and duties as proctologists and to disseminate to the medical profession the knowledge we have acquired in our special field. In a paper read before the Gastro-enterologic-Proctologic Section of the American Medical Association in July, 1946, entitled "Roentgenologic Contribution to the Diagnosis of Functional Intestinal Disorders," Dr. Harry M. Weber of the Mayo Clinic said, "If abnormality is suspected to exist in the rectum or sigmoid colon, the proctosigmoidoscopic examination is done first. This examination has obvious and insuperable advantages over even

the most competently performed roentgenologic examination in the investigation of these portions of the large intestine."

The roentgenologists and the proctologists have learned that a barium enema, in the quest for rectal and lower sigmoidal pathologic symptoms, is the improper procedure, before a digital or instrumental examination has been performed. It is still common practice to request a gastrointestinal series and barium enema for a patient before proctologic study. Suspicion may then arise that warrants the advisability of a proctologic examination. The proctologist's first findings, then, are a bowel wall coated with barium, or, as frequently happens, particularly in·elderly persons, a fecal impaction.

As Dr. Green has demonstrated, there is a very definite place in proctologic practice for the x-ray as a diagnostic aid, particularly air-contrast studies but, like all laboratory aids, it should be utilized as such. X-ray studies above the mid-sigmoid are invaluable, for example, as shown in recent advances made in cecal studies of amebiasis; but caudad to the mid-sigmoid, not only are they of limited value but they may often be misleading.

In view of this, proctologists must stress the words of Dr. Weber which I have quoted. They must stress the simplicity and extent of a proper digital examination, bearing in mind the teachings of that master surgeon-anatomist, John B. Deever, who stated, "The relative positions held by the terminal part of the sigmoid flexure and the first portion of the rectum should be borne in mind, for, with a finger in the rectum quite a small growth in the terminal part of the sigmoid flexure or in the terminal part of the mesosigmoid can be detected, not that the point of the finger is in immediate contact with the growth, but that the tumor can be felt through·the intervening wall of the first portion of the rectum." This is the area that roentgenologically is most confusing and is often referred to as the blind area.

Dr. Green's demonstration has been a lucid exposition of his subject.

ANDREW TAYLOR (Hartford, Conn.): The subject matter of Dr. Green's paper affords very little, if any, opportunity for discussion but it does provoke a few thoughts which I would like to pass on as an appendix to his paper, with his permission. At the risk of exposing an air of repetition, I would like to pass

on these three thoughts which are of prime importance if complete co-operation is to be maintained between the radiologist and the proctologist.

As has been pointed out repeatedly, before an x-ray study is ordered there is a very definite routine from which one should not deviate. This includes a careful history followed by a general physical examination and a thorough proctologic examination.

The latter examination depends upon two pieces of equipment which are always handy, namely, an "educated index finger" and a "seeing eye," augmented by an anoscope and a sigmoidoscope. By the term "educated finger" I refer to a finger which is trained during digital examination to palpate the anal canal and lower rectum and be able to detect differences between the normal and abnormal. By "seeing eye" I mean an observer who knows by sight what normal and abnormal conditions may appear at the distal end of a scope and looks for them.

These steps completed and an x-ray study of the gastrointestinal tract decided upon brings me to my first point: the importance of a proper sequence in the order of study. Due to failure to evaluate symptoms·and findings correctly, one quite often sees a low-grade obstructive lesion of the large bowel precipitated into an acute and complete obstruction by administration unnecessarily of. our opaque meal by mouth. This is not the fault of the radiologist who is merely carrying out such orders as may have been issued for that particular patient. Proper sequence of procedure in the study should have called for a barium enema first which would permit diagnosis of the lesion without obstructing the proximal bowel. A parallel worth mentioning is the fact that even today castor oil is ·still ordered in patients with definite symptoms and signs of acute appendicitis.

A second point I wish to make is that regarding proper cleansing of the bowel before barium enema study. Quite frequently the detection of polypi and even some malignant lesions is difficult under the best conditions. We cannot expect the radiologist to give us the best type of x-ray study if we send him patients for barium enema when proper cleansing has not been accomplished.

My third point concerns the importance of shifts of position of the patient so as to obtain

angle shots of the bowel, especially in the sigmoid region, and spot films.

I am in complete accord with both Dr. Green and Dr. Wagner that there is little if any excuse for an x-ray study of rectum, rectosigmoid and perhaps distal sigmoid because in most patients this can be either palpated or visualized. We are, therefore, imposing a useless task on the radiologist and a needless expense on the patient when we request such a study in an area where we can and should make the diagnosis if we will use that educated finger and seeing eye.

Louis J. Hirschman (Detroit, Mich.): I will dispense with the usual compliments to Dr. Green because whatever he does is always very, very good. I just want to emphasize two other points in connection with this admirable presentation. In the first place, one must remember that the roentgenologist is a consultant and it is the close personal relation between the roentgenologist and proctologist that brings the best results to our patients. I don't believe that we should just send a patient to the roentgenologist simply for films. As often as is possible we should be there ourselves so that we can observe under the fluoroscope the different shifts in positions and the uncovering of lesions that are otherwise overlaid by loops of bowel.

For our information as operating proctologists, we should be able to manipulate the sigmoid so that we know how large or how small a sigmoid we have and how adherent it may be, so that subsequently in the removal of carcinoma or other growths we will know whether and how we can place our colostomy and we will also know whether we will be able to have a perineal implantation with good sphincters which we can prognosticate in advance if we know how much movable sigmoid we have.

Again, in these deplorable cases of women particularly who have had previous abdominal operations and who go from one physician to another for the treatment of so-called colitis, if we would manipulate those cases under the fluoroscope after a barium enema, we would find they have distorted, adhered and angulated colons and that the so-called colitis is merely just colonic dysfunction due to pathologic fixation. We would also save many an embarrassment and be able to cure many patients who now are medical and surgical derelicts.

Frank C. Yeomans (Great Neck, N. Y.): I have very little to add, except that I want to compliment the essayist and the other discussers. I want to say that if you do follow the proper routine, of course, which is the digital, then the sigmoidoscopic examination and you do find a pathologic condition, for example, a carcinoma, that doesn't mean that you are not going to have the x-ray examination. It is very essential to have it, of course. The information derived at that time regarding the length and position of the sigmoid and possible detection of other tumors in other situations may be very advantageous in connection with the treatment. It may determine the type of operation that you will perform in a given case.

G. M. Brown (Bay City, Mich.): I should like particularly to ask a question. I am sure we have all had the experience of having reported a negative barium enema x-ray and find in a few months later that our patient has a carcinoma which perhaps has gone too far. When the roentgenologist goes back and looks at the first film, he says, "Oh, there it was. I didn't see it."

I should like to ask how rapidly do carcinomas grow in the different age groups so we can tell how soon to return the patient for a second x-ray if we are not sure of the first.

Richard Brashear (Columbus, Ohio): I would like to ask Dr. Green if these air-contrast films are of the stereoscopic type or whether it is just one film. I have been able to see in a number of these pictures that have been of the stereoscopic type, polypi which I have missed on the single air-contrast film. Occasionally it will give you an idea as to whether the polyp is in the anterior loop or whether it happens to be in the loop that is posterior.

W. W. Green (closing): I am grateful to the discussants for expressing their views on this subject; their remarks have materially contributed to this presentation.

A point that cannot be too strongly emphasized was made by Dr. Taylor when he warned that the use of barium in the presence of a low grade obstructive lesion might precipitate a complete bowel obstruction.

Dr. Wagner has stressed the superiority of the sigmoidoscopic findings over the x-ray examination in diagnosing lesions in the rectal and rectosigmoid area.

I heartily concur in Dr. Hirschman's statement that a close personal relationship between the roentgenologist and proctologist results in better care of our patients. It is a source

of satisfaction to look up during an operation and observe that the roentgenologist has stepped into the operating room to see the correlation between the actual condition and his preoperative findings. Both Dr. Yeomans and Dr. Hirschman brought out a point not made in this paper. They both discussed the usefulness of information gained in fluoroscopy in determining the choice of the contemplated operative procedure.

The time limitations prevent the answering of Dr. Brown's question in its entirety. In those cases in which there is clinical evidence of a lesion not demonstrated by x-ray the latter examination should be repeated every two or three months at the latest until the cause of the symptoms has been discovered.

Dr. Brashear asked if stereoscopic or single films were employed in the air contrast studies. Although only one plate was used for illustration in this presentation, all patients were examined under the fluoroscope and stereoscopic films were taken.
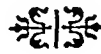
IT is exceedingly important to include sigmoidoscopy in diagnostic study of any gastrointestinal problem. This applies to the child as well as to the adult, and should be a routine procedure regardless of the patient's age. . . . If there is an obscure intestinal abnormality, persistent diarrhea or constipation, rectal bleeding, a discharge of pus or mucus, pain, or any other bowel complaint, a sigmoidoscopy should be performed.

TREATMENT OF COMPLETE PROLAPSE OF THE RECTUM

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Houston, Texas

BEFORE considering the treatment of rectal prolapse, a brief outline of the causes of the disorder should be listed.

It is probable that most men are now in agreement that true rectal prolapse is a

that the lumen of the rectum after prolapse points posteriorly because the prolapsed mass is formed largely at the expense of the anterior rectal wall. Graham[2] gives a good explanation of the mechanics of prolapse. The herniation occurs at the cul-de-sac of

FIG. 1.

Peritoneal incision around rectum

FIG. 2.

Anterior dissection of rectum
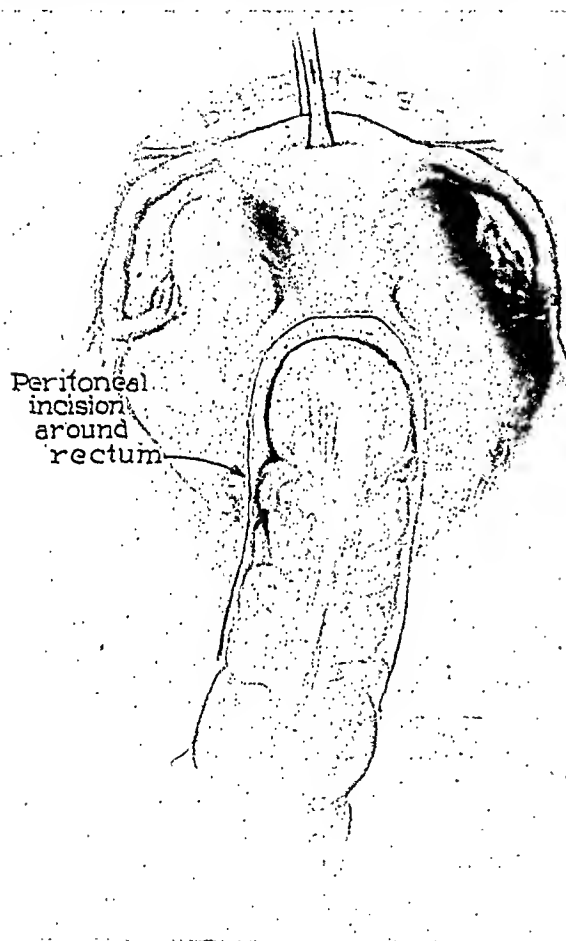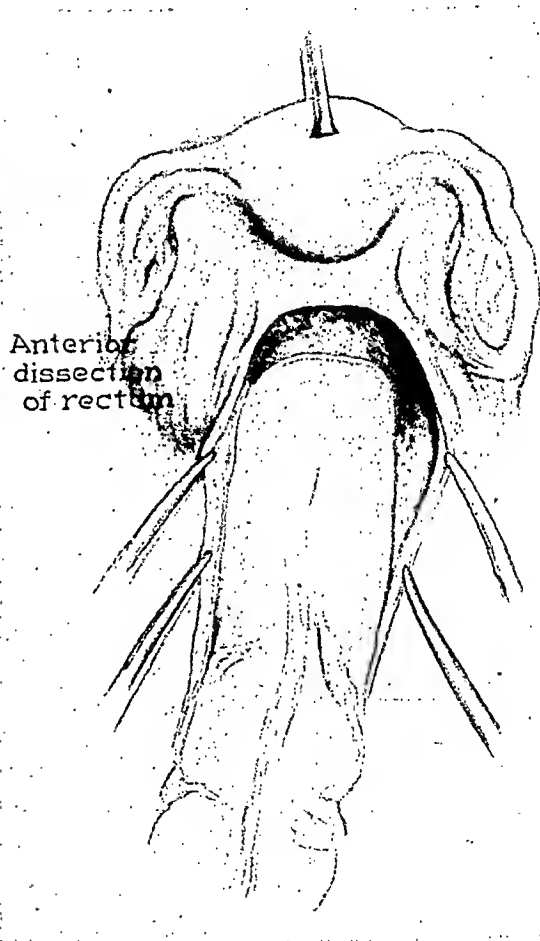
sliding hernia of the anterior wall of the rectum, an idea first advanced by Moschcowitz[1] in 1912. This can easily be proved by pressure of the finger on the anterior wall after reduction of the prolapse which will prevent a recurrence, while pressure on the posterior wall allows the rectum to prolapse immediately. It is also noticeable

Douglas or the rectovesicle pouch and is due to an enlargement of the natural defect in the pelvic fascia that permits the passage of the rectum. The enlargement of this natural defect results from pressure of the contents of the cul-de-sac of Douglas or the rectovesicle pouch into the anterior wall of the rectum, further assisted by such con-

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FIG. 3.

FIG. 4.
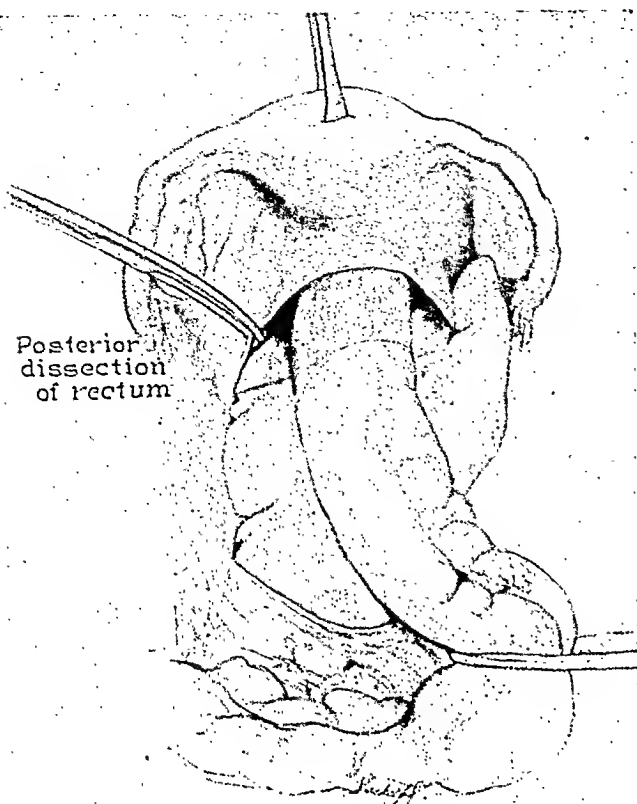
tributing factors as general muscular atony associated with old age, lacerated perineum, chronic wasting diseases with loss of perirectal fat, chronic cough or straining at stool or urination.

The pressure into the anterior rectal wall gradually separates the levators ani, thus permitting enough anterior wall of the rectum to be invaginated into the lumen of the rectum that the latter protrudes through the anal canal. The normal angulation of the rectum becomes obliterated, the posterior wall of the rectum is carried forward from the hollow of the sacrum and the fascial supports become most inefficient due to the stretching. Over the years the mesentery of the small bowel becomes elongated, permitting the latter to lie in the hernial sac and this accounts for the tympanic note on percussing the prolapse.

Several surgical procedures have been advocated for the cure of rectal prolapse. Among these are injection of sclerosing agents, operations designed to narrow the anus and rectum, operations to suspend or fix the prolapsed bowel, operations to restore the pelvic floor, those to resect the prolapsed bowel and others to obliterate the pelvic cul-de-sac. We think that injections of sclerosing agents are sufficient in the cases of smaller recurring rectal prolapse of one or two inches. For the other cases of large recurring prolapse we agree with Graham that adequate treatment should be removal of the sac and repair of the anatomic defect in the pelvic fascia, together with additional fixation of the sigmoid colon.
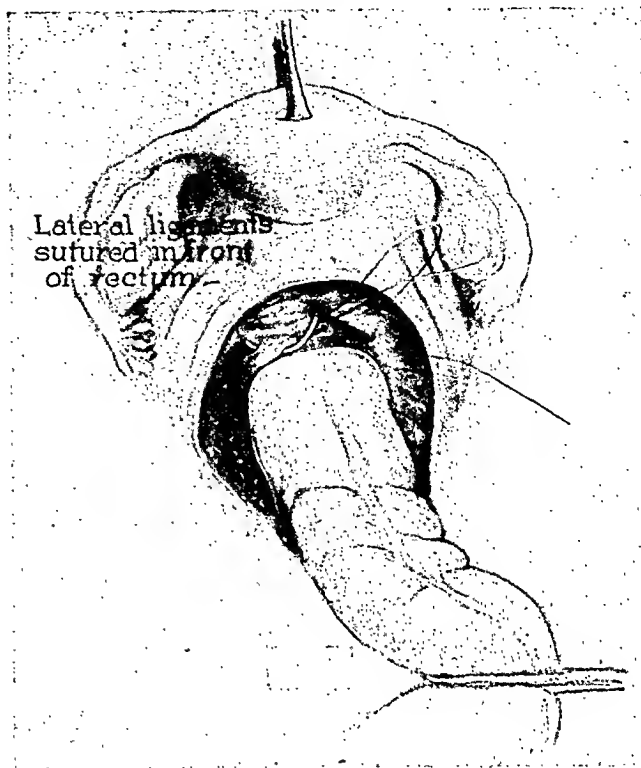
We should like to report nine cases of rectal prolapse seen over a period of as many years. The first three were patients with acute massive prolapse for which no treatment was necessary other than the simple reduction of the prolapse. In each instance it was the first such attack and there was no recurrence. Each patient was a white woman in the fourth decade. One was straining to urinate after eating watermelon and the other two were straining at stool. A good procedure to follow in such cases is to use liberal quantities of toilet tissue when manipulating the mass. Otherwise the slippery surface of a mass the size of a football is most difficult to handle.

Incision of peritoneum lateral to sigmoid →

←Peritoneum sutured
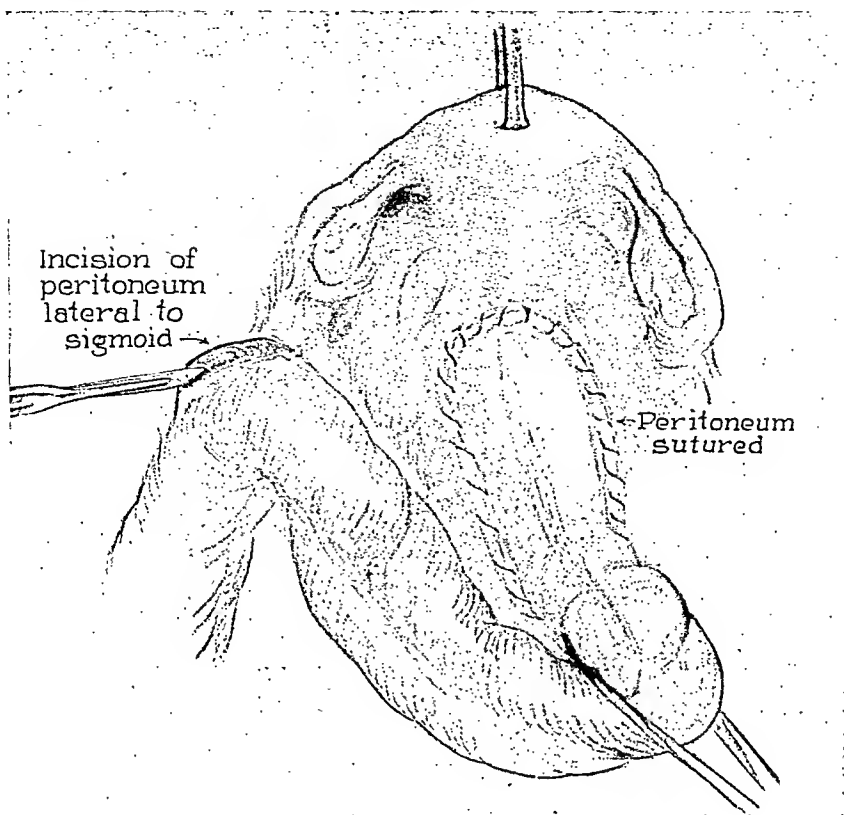
FIG. 5.

Of the remaining six patients, three were injected with sclerosing agents. One boy, age two, was injected with 3 cc. of ethyl alcohol on each side of the rectum above the levators. This was a failure. The other two were white women, thirty-nine and sixty-six respectively who were injected with 1 per cent phenol in castor oil above the levators at each side and posteriorly. Three ounces were injected at each side and one ounce posteriorly. Then, about one ounce of 5 per cent phenol in olive oil was injected under the rectal mucosa, spotting about 5 cc in each of about six sites. Both patients obtained a cure.

The remaining three patients were subjected to abdominal operation. The first was a Negro boy of twenty-two who had a cogenital prolapse of five to 6 inches at each stool. At operation it was found that the rectum was edematous, leathery and thickened so that it filled the entire pelvis. The perirectal tissue was tough, inelastic and edematous and it was technically impossible to properly free the rectum from the sacrum or to expose the pelvic fascia over the levators. All we could do was bring the peritoneum under the rectum as a new pelvic floor. He obtained a partial cure in that only about 2 inches prolapsed after operation.

The last two patients were operated on after the technic of Graham, together with construction of a new bed for the sigmoid which we have added. After a spinal anesthetic an ordinary rectal tube was inserted in the rectum for about 2 inches before the abdomen was opened. Beginning at about the level of the superior hemorrhoidal vessels, the peritoneum on each side of the rectum was incised down to the level of the cul-de-sac and then completely across the cul-de-sac. Finger dissection then freed the rectum from the hollow of the sacrum down to the coccyx just as though removal of the rectum were contemplated. Blunt dissec-

New bed
for sigmoid
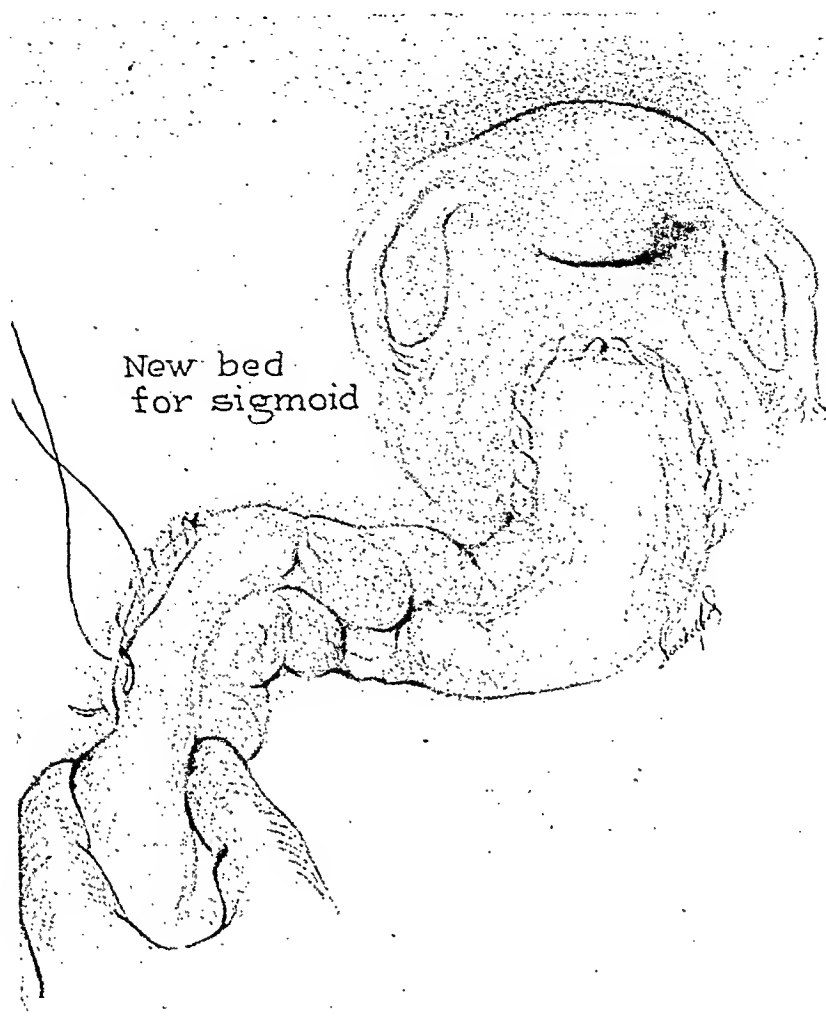
FIG. 6.

tion then freed the rectum anteriorly. The defect between the levators could easily be felt. At this point Graham closed the defect by approximating the levators with sutures through the pelvic fascia over the levators. Both of our patients were males in whom we found this procedure technically impossible. Instead of suturing the levators together we brought the lateral ligaments of the rectum across in front of the rectum and sutured these ligaments together with one or two linen sutures. This took up the slack in the overstretched lateral ligaments and prevented the anterior wall of the rectum from prolapsing through the defect between the levators ani. Then, with the rectum held up taut the cut edges of pelvic peritoneum were turned under and sutured to the rectum at the sides and anteriorly. This obliterated the cul-de-sac of Douglas or the rectovesicle pouch entirely. As a

further support to the repair we made a new bed for the sigmoid by incising the lateral peritoneal leaf from the brim of the pelvis up the lateral gutter for a distance of 4 to 5 inches. After blunt dissection of the thin areola tissue under the sigmoid, the peritoneal leaf was sutured to the wall of the sigmoid while the latter was held up taut. Before closing the abdomen the rectal tube, previously inserted, was pushed up into the rectum and sigmoid by an attendant while being guided by the surgeon's hand. The abdomen was then closed and the protruding end of the rectal tube sutured to the skin over the inner side of the gluteal region. The tube remained for about three days when gas began to pass. By this time the patient was taking liquids by mouth. Mineral oil was then started, one ounce twice a day, the diet increased and the bowels began moving on the fourth

or fifth day. Complete cures resulted in these last two patients and we think that the above procedure is the operation of choice for patients with massive rectal prolapse. The new support of the anterior rectal wall made by suturing the lateral ligaments together in front of the rectum, the obliteration of the pelvic cul-de-sac, and the scar tissue that forms around the rectum in its new bed and the sigmoid in its new bed makes a recurrence virtually impossible.

In none of these patients has it been necessary to tighten or do any plastic work on the sphincter muscles since in each case the muscles were competent.

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DISCUSSION

Thomas Brockman (Greenville, S. C.): A truly significant contribution to the field of geriatrics was made by the late Dr. Granville S. Hanes of Louisville, Ky. Many will remember Dr. Hanes although few were taught his technic. I watched him employ his technic on several patients. Among his patients was one of our own members who, as a result of a Whitehead operation, suffered from a partial prolapse or sagging of the rectal mucosa. Dr. Hanes' technics for treatment of complete and partial prolapse has proved highly successful.

I should like to cite my own experiences with Dr. Hanes' methods. Most of my patients were elderly and their muscular systems had become relaxed, or atonic, as we call it. The majority had borne one or more children. For the most part they were as healthy as may be expected of eighty to eighty-six-year-old women. The difficulty was that when they stood erect for a short period, the anal canal muscles and the rectal muscles would relax and a tubular or globular mass about the size of an ordinary water glass would protrude the entire anal canal and rectum. Naturally, the patients were seriously handicapped even for simple house-

hold duties and exercise, such as collecting eggs, feeding chickens, etc. The patients had been remarkably robust; one was still riding horseback at the age of seventy-five, most of the riding being over the prairies and oil fields of Mexico. It is one of the few gratifications in a physician's practice to encounter such fine patients.

The technic of treatment is somewhat as follows: The patient is placed in a prone position on the operating table with pillows and sand bags under the midriff. The buttocks are retracted by adhesive strips placed on either side. The perianal area is prepared as for surgery by swabbing with merthiolate. The only anesthetic is a very light skin dose of 1 per cent novocain, injected at needle point on two sides. Several ounces of warm, sterilized castor oil is injected perianally. This requires a large No. 16 needle which is introduced along the side of the rectum by guiding its course in the rectum by a gloved finger. A pool of oil is injected on either side or laterally until the surgeon can feel the bulging warm oil mass. The average dose is 90 to 100 cc. of castor oil.

Stimulants are given during the operation and occasionally gas is required. Vigorous finger massage is instituted promptly while the patient is on the table. Heat and massage are continued for one to two hours each day for several days to prevent pooling of the oil and adhesive strips are applied constantly.

The patient requires hospitalization and full time nursing care. Slight shock may be combatted by the usual methods, chiefly stimulants of coramine, oxygen with the rare use of demerol or a very mild hypodermic of pantopon. The chief weapons for after-care are heat and massage and in the later stages the patient appreciates hot sitz baths.

I have never encountered a recurrence. The age of the patient may be a factor in this observation. Nevertheless, the remaining years of the elderly woman are made comfortable and happy.

Rufus C. Alley (Lexington, Ky.): It is a pleasure to discuss such a scientific and well presented paper.

The multiplicity of treatments, operative and otherwise, which have been advocated for rectal prolapse indicate that no single method is ideal or universally applicable. This point is illustrated by the fact that Drs. Hayes and Burr used three different methods in the treatment of

nine patients: (1) simple reduction; (2) sclerosing injections and (3) surgical repair.

Their explanation of the mechanism of complete prolapse is perfectly logical as is their method of surgical correction of the responsible anatomic defect.

The great majority of patients with rectal prolapse that I see are either young children or elderly debilitated individuals. In my opinion a child with rectal prolapse, either partial or complete, should not be subjected to surgery until all simpler corrective procedures have been tried and have failed. Co-operative management by proctologist and pediatrician will, in most instances, lead to spontaneous cure in these cases. The reason for this is because as the child grows the rectal bed increases in length more rapidly than does the rectum, thus taking up the slack which was responsible for the prolapse.

The elderly debilitated patient with rectal prolapse is an entirely different problem. In most of these patients spontaneous improvement is not to be expected and because they are poor surgical risks major surgery is not feasible. In this type of patient the judicious use of sclerosing injections is the safest procedure for palliation.

E. G. MARTIN (Detroit, Mich.): I would like to restrict my discussion to the paper as presented which I enjoyed very much. I would like to accentuate what the Doctor has reported, that after he has done what he has disclosed in his drawings, the patient will not have a prolapse again; it would be practically impossible.

The first thing that happens is prolapse of the sigmoid into the rectum, a colonic rectal prolapse. This has been classified over the years as third degree. Since this is the first thing that happens, I classify it as first degree.

Patients who have their two ligaments, one of which is at the junction of the pelvic colon and rectum and the other at the pelvic colon and iliac colon, can have prolapse if those ligaments are not normal. I do not know about the defect of the anterior part, but I do know from rather long experience in quite a number of patients that if you put your hand in the abdomen when you have it opened and pull up the gut, it will not recur as long as you hold your hand there. It cannot recur, because it cannot get away from your hand. We simply fasten the gut to the iliac fascia or the psoas spina muscle, and about five or six plain catgut su-

tures cover up the raw end of it and we can omit all of this dissection and prolongation of this operation which Dr. Burr has so well presented. Another prolapse probably will not occur after the gut has been well fastened; at least, in our experience we have never seen or heard of a recurrence of prolapse after this treatment.

EMMETT H. TERRELL (Richmond, Va.): I would like to discuss rather briefly the method which we use in the treatment of prolapse in children. A distinction should be made between simple incomplete and complete varieties of prolapse of the rectum. In children almost invariably it is simple prolapse of the mucous membrane.

Prolapses occurring in children are not necessarily confined to those that are debilitated but we find it very frequently in well nourished and otherwise perfectly healthy children. For the last twenty-five or thirty years, we have simply been treating these prolapses in children by the injection of quinine and urea hydrochloride, either 2 or 3 per cent, after the same method that you use in the treatment of simple, uncomplicated hemorrhoids. I cannot say how many children we have treated during this period, but it has been quite a large number. In most instances the symptoms are entirely relieved after the first treatment. In a few cases when you have had to repeat the treatment for as many as four or five times, there has not been a failure to completely relieve the symptoms. As far as I know, there have been no recurrences, after this method of treatment. Of course, it would not apply if the muscular parts of the bowel were prolapsed. This treatment is only applicable in those cases in which the mucous membrane protrudes.

RUDOLPH V. GORSCH (Flushing, N. Y.): A great deal has been written about injection therapy in treating prolapse. Obviously, it is not always a satisfactory method. There is a very important technical point I would like to bring out about it. I remember years ago we brought Dr. Hayes up to New York to see how he conducted the injection method. Having used it beforehand, I learned very forcibly at that time that we were not using the proper technic. In using the injection method, whether you use quinine urea, castor oil or hydrochloric acid, it is essential that you put your solution directly along the inner edge of the bowel wall because I think ultimately your success upon

holding up the prolapse depends upon the amount of fibrosis you get. You absolutely have to flood the solution right alongside the bowel wall. If you put it out a centimeter or a centimeter or two, obviously you don't get any fibrosis or fibrous fasciculi which will eventually hold the bowel wall up.

I think that is a very important point in the injection treatment.

RAYMOND L. MURDOCH (Oklahoma City, Okla.): There is a great tendency when a paper has been published, particularly one with excellent diagrams, as Moschcowitz paper had, subsequently to say that all prolapses are caused by defect in the fascia of the anterior wall and that the anterior wall of the rectum is the part that is the initial part of the prolapse. But I do not believe that all prolapses that appear on our hospital records are prolapses through the cul-de-sac. I feel that the great majority of them are just a little more severe or extensive processes of ordinary hemorrhoids, the prolapsing of large hemorrhoids with a little additional severity of that process and then diagnosis in hospital records appears as prolapse. So the thing that must be done in every case is to feel the prolapse all around as well as look at it and percuss it, in order to detect a tympanic note although I have found this very difficult to elicit. I have been very much in doubt as a matter of fact whether only the mucous membrane prolapsed or whether all coats of the bowel prolapsed in the patients I have had.

Therefore, I think it important not merely to accept Moschcowitz's beautiful diagrams, but to take each individual case, inspect it very carefully and particularly feel it.

The great majority of patients who are diagnosed as having prolapse show a mass out at the anus. This is a prolapse of the anus rather than a prolapse of the rectum. Next time determine the amount of sulcus. Start in at the anal border and see whether the finger will extend into a fairly normal anus before it strikes this intussuscepting mass. In other words, see how much sulcus there is there before you get the prolapse.

The next method is the Moschcowitz diagram procedure. In females it is unnecessary to go into the abdomen to remove and correct the prolapse. The abnormal mass can be removed by going through the rectal vaginal septum. It is a common practice for correcting defects there.

The very rare case is the one in which it is necessary to go into the abdomen to make a fixation of the colon. If this canal is narrowed and the defect repaired which can always be done in the female from below, then it will seldom be required to go into the abdomen for operative procedures on prolapse in females.

HARRY B. BURR (closing): I would like to thank the discussers for their kind and generous remarks. It is a healthy thing to have a difference of opinion.

I agree with Dr. Alley, of course, that in treating old people, one should not subject them to abdominal operations. I do not believe in removing a prolapse of the bowel from below. I think it is dangerous because frequently loops of small bowel are in the prolapsed mass. I am speaking of the massive prolapse, not the small prolapse of the mucous membrane.

DIAGNOSIS AND TREATMENT OF PAPILLARY ADENOMAS OF THE RECTUM

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THE most common tumors found in the rectum are those which arise from the mucosal glands and are classified as adenomatous tumors. These tumors in accordance with the histologic appearance may be divided into two main groups, benign and malignant. Benign tumors, regardless of shape and size, are often termed polyps, while the malignant variety are known as malignant polyps or rectal cancer. Differentiation between the benign and malignant group, with recognition of the characteristics of each, is essential if patients are to receive the most appropriate treatment.

There are three clinical types of benign adenomatous tumors: (1) areas of hyperplasia, (2) adenomas and (3) papillary adenomas.

Areas of hyperplasia are small, sessile, slightly raised tumors which occur most frequently in the mucosa above or below rectal cancer and also on mucosa which has been exposed to irritation for a comparatively long period, e.g., the surface of a colostomy. It is possible that areas of hyperplasia are at times forerunners of more mature adenomatous tumors.

Microscopically, areas of hyperplasia show only slight differentiation from the surrounding mucosa. The glands are somewhat larger and contain cells which are slightly elongated and more closely packed. The nuclei may be hyperchromatic. However, the orderly arrangement of the mucosal glands is nowhere lost and blood-vessel distribution is identical with the adjacent normal areas.

Adenomas are the most common of the three benign varieties. These polyps are usually small or of medium size. They may be single or multiple, smooth or ulcerated,

sessile or attached by a pedicle. The induration is usually sufficient for recognition by palpation with the finger.

Adenomas are composed of arborescent masses of large proliferative glands upon an orderly frame-work of connective tissue which radiate from a central fibrous stalk in which the blood supply is carried and which is developed from the submucosa. The stalk may be elongated to form a pedicle, in which case it is covered by normal appearing mucosa. The glands are much larger than normal, both in length and diameter, and vary in degree of mucus secretion. The cells of the glands are generally closely packed and elongated, and their nuclei are compressed and hyperchromatic. Mitoses are frequently seen and the cells are sometimes multilayered.

Papillary adenomas are seen less commonly than the other two groups. They generally occur in an older age group than the adenomas and are usually single and sessile; when they are large, they have overhanging margins. A pedicle, if present, may be almost equal in diameter to the tumor itself. The base of the polyp is frequently circular but often becomes quite irregular as the size of the polyp increases. The thickness varies widely in different cases and also in the same tumor. One area of the tumor may be a millimeter while another area of the same tumor is several centimeters in thickness. The surface presents a papillary or in some cases a lobulated appearance which is characteristic of this type of disease. Papillary adenomas are soft and spongy and it is not unusual for them to be missed on rectal palpation.

Papillary adenomas are similar in microscopic appearance to adenomas; in addition the greater part of the surface is composed

FIG. 1. Shows early secondary gland formation or atypical change in a villus.

of thin villous processes covered by single or multiple layers of elongated columnar cells upon a basement membrane supported by a delicate fibrous tissue core which carries the blood supply. Small papillary adenomas develop from a single central fibrous stalk, but the majority of them are composed of multiple stalks springing sometimes from a wide area, each stalk supporting a papillary aborization. Infiltration of eosinophiles, plasma cells and lymphocytes are frequently seen in the central stalks and their prolongations. Also, it is not uncommon to find interstitial edema of the individual villous process. The degree of mucous secretion can vary markedly in different areas of the same tumor. Mitoses are frequent and not considered significant.

Atypical changes may be present in the papillary adenomas, with or without areas of outright malignant transformation. These changes consist of focal or diffuse areas of papillary tufting of epithelial cells into the lumen of the glands or onto the surface of the villi without disruption of the basement membrane. In some cases there may be diffuse atypy throughout the specimen. In others, there is a relatively benign structure throughout the greater part of the specimen, with focal malignant areas without infiltrating qualities, as evidenced by distortion of gland structures and divergence in size and shape of the nuclei with apolarity. Others will show a diffuse atypical structure with small focal areas of frank carcinoma infiltrating within the polyp. Many degenerative changes simulating malignancy may be seen in the tips of the villi. Also, secondary villi may be found springing from within the lumen of glands.

Papillary adenomas may be separated for convenience according to their clinical and microscopic appearance into four groups: (1) tumors that are clinically and microscopically benign; (2) tumors which show microscopic atypical change; (3) papillary adenomas in which there are islands of adenocarcinoma and (4) fully developed infiltrating carcinoma in which areas of benign papillary adenoma still remain.

One cannot overemphasize the importance of recognizing clinically the characteristics of the benign, atypical and malignant types of papillary adenomas. Classification may be impossible by any

FIG. 2. Shows more advanced secondary gland formation or atypical change in a villus with moderate papillary tufting of surface epithelium.

other method prior to complete surgical removal. Due to the previously mentioned wide microscopic variations, repeated biopsies often fail to reveal existing atypical or malignant features. Factors to be considered in estimating malignant probabilities are induration, ulceration, color and bleeding or oozing following moderate trauma to the polyp.

The small or medium-sized purely benign lesions are soft and about the same color as the adjacent mucosa. They are not ulcerated and do not bleed readily. Benign polyps revealing atypical changes will show a slightly increased vascular appearance to the above, bleed or ooze slightly to trauma. All polyps which have undergone definite malignant change have a recognizable degree of duration and sufficient blood supply to produce considerable oozing or bleeding when traumatized and after taking a biopsy. It is not unusual to find superficial ulceration in a percentage of malignant cases.

Determination of the appropriate form of treatment is at times a difficult problem. Factors worthy of greatest consideration are the previously described clinical characteristics, together with the size of base and extent of involvement of the rectal wall, and the presence or absence of induration at the base. The objective in all cases is complete eradication of the tumor with a minimum amount of morbidity and rectal deformity.

For descriptive purposes treatment may be divided into steps or stages: A number of the smaller papillary adenomas are eradicated by the initial procedure while the more extensive adenomas require additional treatment. Partial or complete removal of all papillomatous tissue is attempted in the first stage. Removal may be by means of an electric snare or electric snare together with fulguration of the base of the polyp. In certain cases local surgical removal by dissection and suture is the method of choice. Methods which permit histologic study of the tumor tissue removed are preferable to those which destroy it.

The second step of treatment consists of radiation therapy. It is reserved for patients in whom snare and fulguration or local surgical removal have failed to eradicate the disease completely. Radiation

therapy consists of external applications of high voltage roentgen rays, surface applications of radium and interstitial implantations of gold-filtered radon seeds.

External applications of high voltage roentgen rays are administered through six or seven pelvic portals, the rays being focused at the center of the lesion. Surface applications of radium are administered by means of a rectal applicator which holds the radium at a distance of 10. to 1.5 cm. from the surface of the polyp. Small daily dosages delivered in this manner produce a favorable effect upon the tumor without causing any unfavorable reaction to the patient. Islands of polypoid tissue which remain after moderate dosages of radium have been administered by rectal applicator, may be treated by implantation of gold filtered radon seeds. It is our impression that snare removal, superficial fulguration and radiation therapy is followed by less deformity of the rectum than that following focal surgical removal of a large sessile tumor.

Radical surgical removal of the rectum constitutes the third step of treatment. This radical treatment is reserved for those conditions which have not been cured or controlled to a satisfactory degree by the above less radical methods. Tumors which show induration at the base and those with fully developed papillary adenocarcinoma should be treated in this manner.

All patients who have received conservative treatment for papillary adenomas warrant careful post-treatment observation. There is marked tendency for these tumors to recur. Recurrence may be either early or late. Moreover, even with an initially benign form of tumor, recurrences may reveal atypical or malignant cells. Because of the frequency of carcinomatous change in the primary and recurrent tumors, all patients must be carefully watched and the lesions considered to be potentially malignant.

DIAGNOSIS AND TREATMENT OF MUCOSAL POLYPS OF THE RECTUM AND COLON, WITH EARLY MALIGNANT CHANGE*

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THERE are certain conclusions which are generally accepted at the present time concerning mucosal polyps of the rectum and colon: (1) Polyps of the terminal bowel, the majority of which are adenomatous in character, are true tumors and not the result of inflammatory processes. Clinically, they must be considered premalignant lesions.[1] (2) The incidence of mucosal polyps in the adult population is much greater than has previously been suspected. Hang[2] and I in a series of 1,843 autopsies performed at the Pathological Laboratory of the New England Deaconess Hospital found 311 patients with benign mucosal polyps, an incidence of 7 per cent. Forty-two per cent of these patients had multiple lesions. Helwig[3] more recently has reported an incidence of 9.5 per cent in a large autopsy series. (3) The detection of large numbers of colon and rectal polyps has been difficult. Only a small percentage of such patients complain of rectal bleeding, and a still smaller group is found to have polyps which are benign and yet have attained sufficient size to cause obstructive or other bowel symptoms.

It has been demonstrated that over 50 per cent of the benign polyps of the terminal bowel are within reach of the 10-inch sigmoidoscope,[2] but the radiologic detection of small tumors beyond the reach of the sigmoidoscope is unsatisfactory. Only a small number of the mucosal polyps of the right colon, transverse colon, descending colon and upper sigmoid, which we believe to be present, are detected by the usual barium enema radiographic studies of the colon or by the contrast air technic. Because of the high percentage of these tumors within reach of the 10-inch instrument, however, as we have broadened our indications for sigmoidoscopic examinations we have discovered more and more mucosal polyps.

To discover the greatest possible number of premalignant lesions of the colon and rectum, a sigmoidoscopic examination should be carried out on all patients over thirty-five years of age as a part of a general physical examination. Internists, surgeons and all physicians doing general diagnostic work should be prepared to do examinations of this nature. It requires a minimum of experience to learn to pass a sigmoidoscope and to determine the presence or absence of a tumor. Once a tumor has been discovered, however, it may require the highest degree of skill and experience to determine the presence, degree and extent of any malignant change which may be present and the type of treatment indicated.

It is the purpose in this presentation to review the criteria for the diagnosis of cancer in colon and rectal polyps, and to discuss the treatment indicated when malignancy is found. Also, a series of twenty-two cases will be reported in which the diagnosis of malignancy was made sufficiently early so that local excision or destruction of the polyp was adequate to control the disease.

This series of twenty-two patients is admittedly a small number as compared with the number of patients seen at the clinic with cancer and benign tumors of the colon and rectum. Over 2,000 patients have now

FIG. 1. An early carcinoma in a mucosal polyp; tumor removal by local excision. × 150.

FIG. 2. A small focus of carcinoma in a mucosal polyp; tumor removed by local excision. × 150.

been operated upon for malignancy of the large bowel and several hundred additional patients with benign polyps of the colon and rectum. Seventy-six histologic specimens of benign polyps of the colon and 684 of the rectum have been studied in addition to the large number of patients who have had small mucosal polyps fulgurated through a sigmoidoscope without biopsy specimens being removed. As more and more polyps are discovered, the problem of the early diagnosis of malignant change in such tumors with the possibility of avoiding radical resection of the bowel in a small number of such patients will become increasingly significant.

In the detection of malignancy the value of palpation of these tumors cannot be overemphasized. The soft, pulpy, non-indurated, freely movable polyp discovered in the ampulla of the rectum will rarely be found to be cancer. Induration, fixation and ulceration to the examining finger,

however, are almost always pathognomonic of malignancy. The visualization of these tumors is less important than palpation or histologic study. To the trained observer, ulceration and defects in the usual normal-appearing mucosal surface of these tumors are significant. The typical ulcer of malignant disease, with firm, rolled edges is diagnostic.

The final proof of malignant change in a polyp depends on histologic study, not only of biopsy specimens of the polyp but of representative sections of the entire tumor, together with its stalk and attachment to the bowel wall as malignant change may develop in any portion. It has not been an infrequent experience to have suspected cancer in a polyp because of firmness or induration to palpation and only after a large number of biopsy specimens have been taken from various sections of the tumor could malignancy be demonstrated.

Histologically, it may be relatively difficult to determine the presence or absence of malignant change in many intestinal polyps. Frequently patients are referred to the clinic for a final diagnosis where our pathologists disagree with previously reported findings. It is essential in these borderline cases that a pathologist experienced in this field be available. Warren[1] and I in a paper on this subject have stated that "If we accept three important criteria of malignancy—anaplasia, irregularity of architecture and invasion—it is necessary to have at least two of these three factors present before making a diagnosis of malignancy. It is possible for any one of these three criteria to be present without actual malignancy, with one exception. Definite lymphatic or intravascular invasion means clinical cancer."

These criteria from a histologic standpoint have been followed in our patients for a number of years and have been highly accurate in the determination of actual cancer.

When palpation reveals a freely movable, soft, non-indurated tumor and it is possible to demonstrate histologically that there is no malignancy in any section of a polyp, its stalk or adjacent bowel wall, local excision or complete fulguration is indicated. These patients should be studied for additional polyps because of the high frequency of the multiplicity of these tumors and, probably because of the inherent growth defect in these patients, they should be examined at intervals indefinitely.

When palpation of such a tumor reveals induration, ulceration and fixation of the tumor and a biopsy specimen taken from the periphery shows malignant change, radical resection is the only procedure indicated.

When palpation reveals induration, ulceration and fixation of a polyp and such a biopsy does not reveal malignant disease, the final diagnosis should be deferred until repeated histologic studies have been made of all sections of the tumor together with its stalk and adjacent bowel wall; and only

FIG. 3. Multiple polyps in sigmoid area.

then, with such histologic findings interpreted by an experienced pathologist, can cancer be ruled out.

When palpation reveals a freely movable, soft, non-indurated tumor and histologic studies disclose a polyp with early malignant change confined to a single area and not involving the stalk or adjacent bowel wall, then, and only then, should local excision or destruction of the tumor be considered.

In a review of the twenty-two cases reported in this series, certain additional data are of significance. Twelve of the patients who were treated by local excision or destruction of the tumor had polyps showing local, early malignant change within reach of a 10-inch sigmoidoscope. Two patients had a subsequent radical resection because of a suspected involvement of adjacent tissue by the malignant process but no malignant change was found in the removed specimens. Bleeding was noted by all of these patients. Age and sex factors were not significant.

Ten of these patients had tumors in the colon, eight in the sigmoid area and two in the descending colon. Forty-one patients

FIG. 4. Large benign mucosal polyps in sigmoid area.

FIG. 5. Large fecalith mistaken for tumor.

have been operated upon at the clinic by colotomy with local excision of the polyp. In this group, histologic study revealed no malignant change in thirty-one patients; in twenty-eight the polyp was in the sigmoid area, in one patient in the transverse colon and in two patients in the descending colon. Ten patients had polyps with localized early malignant changes; eight were in the sigmoid area and two were in the descending colon. Two of the patients with early malignant polyps later had resection because of the extent of the tumor but no residual cancer was found. Bleeding had been noted by thirty-eight of these forty-one patients. There was one postoperative death, a mortality of 2.4 per cent.

It is to be noted that the incidence of early malignant change in those polyps found above the reach of the 10-inch instrument is many times greater than in those found below this level. The incidence in this series was 24 per cent. This finding is probably explained by the fact that few

tumors are discovered in the colon until they have reached a centimeter or more in diameter, have been present for an appreciable period of time and may have developed peripheral ulceration with bleeding resulting from their beginning malignant change, which symptom originated the investigation during which the tumor was discovered.

In conclusion, twenty-two patients are reported with mucosal polyps of the rectum and colon having early malignant change, treated by local excision, and followed for varying intervals of time up to seven years with no recurrences. It is to be emphasized that no attempt is made in this presentation to advocate anything but the most radical type of surgical procedure for clinical cancer of the colon and rectum. It is to be hoped, however, that as the medical profession becomes increasingly aware of the significance and frequency of the premalignant lesions of the colon and rectum, not only will an increasing number

of patients be prevented from developing cancer of the terminal bowel by the discovery and destruction of these tumors in this premalignant state but there will be an appreciable number of patients having polyps showing early malignant change who may be safely treated conservatively.

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DISCUSSION OF PAPERS OF DR. BINKLEY, DR. SUNDERLAND AND DR. SWINTON

D. R. LAIRD (Portland, Ore.): It is a pleasure to discuss these interesting papers but difficult in that they are so complete and there is so much agreement in the main.

The division of benign adenopapillomatous tumors into three clinical types with correlation of the clinical characteristics and histologic appearance is of value and I agree entirely with Dr. Binkley's observations.

It should be emphasized again that the clinical characteristics mentioned by Dr. Binkley, i.e., induration, ulceration, fixation and vascularity certainly are as important as the microscopic findings of biopsy in determining the type of treatment. I agree that even multiple biopsies may fail to reveal the presence of malignancy and that when possible the entire lesion should be removed in one operation so that complete histologic study may be made.

Dr. Binkley's division of papillary adenomata into four groups for the purpose of clarifying treatment is advantageous. There is no doubt that the decision as to what type of treatment is to be used is at times difficult and should be made by one having great interest and experience in this field. So many variables are present that hard, fast rules cannot be made to cover individual situations. As a guide we should all ask ourselves this question when confronted with such a decision, "What would I choose to have done if I were the patient"? This helps to bring the problem home in a real way.

In regard to treatment of these lesions, fulguration, snare excision and local excisions to remove the entire thickness of the rectal wall all have their place in clinically benign to border line lesions. The principle of fitting the operation to the patient and not the patient to the operation pay dividends in reduced mortality and morbidity. This is illustrated by the unusual circumstances involving a patient in which two previous attempts elsewhere, over a two-year-period at destruction of adenopapillomatous hyperplasia of the entire rectal ampulla, had failed. When seen, the patient was adamant in refusing any type of abdominal operation. The lower 10 cm. of her rectal mucosa was diffusely involved by adenomatous hyperplasia varying from 1 to 3 cm. in thickness. Fulguration was considered hesitatingly because of the huge area involved but, when it was discovered that the rectosigmoid was mobile and could be brought down to the anus easily, the entire rectal ampulla, wall and all was dissected free and the rectosigmoid sutured to the sphincters. This patient left the hospital on the eighth postoperative day with excellent bowel habit and control and has remained so for the past eighteen months. This same procedure has been used on three other occasions, once for recurrence of carcinoma in a stricture following fulguration in a ninety-four year old patient and twice for bladder and prostatic rectal fistulas which were so extensive that they approached the nature of a cloaca. The results were equally good.

In addition to the necessity for continued observation of these patients, I should like to stress one point not previously mentioned. That is the extreme importance of excision of these lesions without trauma. Metastasis can occur at time of removal if the lesion is squeezed or clamped and in clinically malignant lesions it is wise to block the blood and lymph return before proceeding with difficult excisions.

Continued reexaminations of patients, even with obviously benign lesions, are important and the patient can be made to realize this without being frightened or made neurotic. In event of recurrence it is frequently not too late for more radical procedures aimed at really curing the patient.

The only point of disagreement with Dr. Binkley is in the use of x-ray and radium. My experience has been limited, admittedly due to my feeling that if I were the patient and had to

depend on radiation to eradicate cells growing beyond the reach of local excision or fulguration, I fear I should not sleep well. I need more convincing evidence that x-ray does anything to cells of colonic origin, especially when given via the usual portals.

In Dr. Swinton's thirty-five patients with adenopapillomatous lesions showing early malignancy which had not yet extended into the base of the lesion, he had no failures when the patients were treated with local excision. I have not been so fortunate. Within one year after local excision of such a lesion, an extrasigmoidal mass could be noted and I had to do a combined abdominoperineal excision at which time it was found that the only metastasis was about 4 to 6 inches away from the bowel which had apparently involved a lymph node.

W. J. Martin, Jr. (Louisville, Ky.): These have been two very comprehensive papers which I think will be a great addition to the literature. I think the discovery and eradication of these growths is more or less vital to the patient, because I think it is becoming generally accepted these things are definite precursors of cancer; and if we are careful in our examination and as we extend the number of examinations and the indications for the examinations, which has been brought out, we will find at least in some of these patients whom we can get to early that the eradication is not apt to be so radical.

In my series of cases I have found that approximately 7 per cent of the people that I examined have some type of papillary adenomatous growth in their bowel within reach of the sigmoidoscope. I do not believe that the gross recognition in determining whether or not these things are cancer or whether they are benign is very accurate. I think most of them that I find are above the finger, where you cannot palpate most of them. I think that the rectosigmoid growth is situated so that you cannot reach that high and do a good palpation on it in most cases. The gross appearance of them is very deceiving at times. I do not believe that too much stress should be placed on that. I think that microscopic diagnosis is the criterion. Of course, if there is a grossly ulcerated mass there in which all polypoid characteristics have been lost, that is a different story. These things usually occur singly, but with the help of the double contrast enema which is always used and always should be used if we find one, occasionally we find other growths higher up, which can be watched if one chooses to do that for a while, or radical surgery could be instituted at that time.

I think that in treating these things one has to be very cautious. It would be very nice if we could remove all of these by snare or fulguration and save people the risk of major surgery, but I think it should be emphasized to a group such as this that one should be very, very careful in choosing this type of treatment. The removal by snare and fulguration can be done by most anyone if he is cautious so that he will not run into bleeding; but I think if there is any one thing that we should carry away from here it is that the use of the fulguration machine and the snare should not be overemphasized.

Speaking of recurrences of these growths, sometimes I wonder if they are real, true recurrences. Why not a new polyp which has developed? Very frequently if, as I say, there is more than one and you remove the specimen surgically, you find small polyps adjacent or close to the original growth. It might be if one has done a local resection or has used the snare or fulgurating tip and removed a growth, it is perfectly possible that a small polyp might have been overlooked at the time and that the recurrence does not turn out to be a recurrence but an entirely new growth.

I believe that the age group has something to do with your choice. As one man brought out on prolapse of the rectum in an earlier paper, in these older people recurrence does not occur. I do not suppose their life expectancy was great enough to give them time for it to recur. I think in some of these individuals that life expectancy is short enough so that perhaps you might be a little more lenient and probably do a fulguration on some of those in the older group.

A. W. Martin Marino (Brooklyn, N. Y.): Tumors of the rectum are either benign or malignant. Benign tumors include fibromas, lipomas, angiomas, myomas and adenomas. The last named are the predominant type. Hyperplasia is not a form of neoplasm and should not be included in a discussion of neoplasms except from the standpoint of differential diagnosis. However, I might say at this point that any thickening or protrusion of the rectal mucosa demands careful investigation.

At times the term adenoma is loosely used as though some were benign and some malignant. When malignant transformation is proven, the lesion is no longer an adenoma but a carcinoma. Although adenomas are benign tumors, it should be kept in mind that they are premalignant lesions and therefore very important from the standpoint of early discovery as well as prompt and effective treatment. It is well to keep in mind, too, that glandular tumors of the large intestine may be multiple and for that reason the finding of an apparently isolated lesion in the rectum should make the examiner suspect the presence of other tumors, benign or malignant, higher up.

After I have become convinced that I am dealing with a solitary or with multiple papillary adenomas, all of which are within reach of the protoscope and amenable to local eradication, the treatment I employ is to remove the tumor or tumors *in toto* by means of the electrocoagulation snare with fulguration of the base. The removal of the lesion or lesions is accomplished in such a way as not to distort the specimen or specimens. To quote our pathologist at the Brooklyn Hospital, Dr. J. Arnold deVeer, "By all means have all tissue that is removed examined and the method of removal should be, if possible, the one that least interferes with histologic examination." I make it a rule to remove the tumor or tumors intact and not take a biopsy. Two things are accomplished by this procedure: first, the intact tumor permits serial microscopic study of the entire tumor and second, if the tumor proves to be benign and its removal has been properly accomplished and we are reasonably certain of its complete eradication, then the only thing that need concern us is the possibility of the development of a new lesion. For that reason we insist upon periodic check-ups of any patient who has been treated for an apparently isolated lesion.

I do not see any reason for removing a piece of a papillary adenoma for microscopic study when it is just as easy and certainly more satisfactory, so far as I am concerned, to remove the whole tumor and at the same time perhaps cure the patient all in one stage. In this connection I want to emphasize what Dr. Binkley said concerning the importance of estimating the malignant possibilities of the lesions under discussion by noting their clinical characteristics such as color, induration, ulceration and

hemorrhagic tendencies. Such an estimation is a guide as to the form of treatment to be used. The final diagnosis, of course, is made by the pathologist.

In removing these small tumors through a proctoscope, general anesthesia is not needed because the cooperation of the patient is desired. Special instruments are essential: a good light, suction to remove smoke and fluid, snares and electrodes on long carriers, a reliable diathermy machine and a long forcep to recover the tumor after its detachment. Skill in the use of the proctoscope and experience in electrosurgery are prerequisites.

In certain fields of surgery, such as those dealing with the larynx and cervix uteri, an attempt has been made to recognize malignancy in epithelial membranes before there is frank invasion of the lamina propria or deeper layers. These lesions have been called "carcinoma *in situ*." In the case of rectal neoplasms, the criterion for recognition of malignancy should perhaps also be *not* actual invasion but early evidence of anaplasia prior to the invasion. In our hospital when the lesion is reported as malignant, a distinction is made in these relatively small tumors as to whether they are invasive or malignancies *in situ*. If it is a malignancy *in situ*, removal by electrocoagulation with fulguration of the base suffices in my experience. On the other hand, if the specimen exhibits invasion of the submucosa, I perform radical resection because that makes abdominal exploration for metastases possible and, furthermore, only by microscopic examination of all the coats of the bowel wall can I be certain of the extent of invasion of its coats.

In conclusion I would say that I divide rectal tumors into two classes: those that are benign and which can be cured by local eradication and those which are malignant. I divide the malignant tumors also into two classes: those that are malignant *in situ* and amenable to local eradication and those that require the more formidable forms of treatment. So far as I am concerned, prompt, adequate and radical surgery is our best weapon against rectal carcinoma, especially early carcinoma.

R. A. SCARBOROUGH (San Francisco, Calif.): I think very much we need a pathologist here for this symposium discussion. We are using terms more or less loosely. I think in some of the discussion we have missed the significance of Dr. Binkley's first paragraph, in which he

attempted to isolate one particular group of adenomatous tumors and classify them as papillary adenomas. All three discussants used the term "papillary adenoma" more or less generally, but this is a particular group of adenomas which give us the greatest concern. They are the most treacherous type of tumor which we have to deal with in the colon in making the decision as to what kind of a tumor it is.

Dr. Marino has classified his tumors as benign and malignant. I certainly think he needs a third classification of "or both," because in the majority of the papillary tumors which Dr. Binkley has described, biopsy will usually reveal the appearance of a benign tumor. In this particular type of tumor malignant change develops in the base of the tumor usually centrally located, hard to approach for biopsy, whereas in the ordinary adenomatous polyp which becomes malignant, degeneration occurs on the periphery and is subject to approach by superficial biopsy. Size in papillary adenomas has no direct relationship to the possibility of malignant degeneration in the base or the degree of malignancy present. We need not only a pathologist in helping us to decide about these tumors but I think we need a pathologic specialist. There are too many pathologists today in this country, certainly in my part of the country, who facilitate their diagnosis by calling every adenomatous polyp of the rectum or colon carcinoma grade I. I do not believe that helps us at all in our decision as to what to do for those patients.

We have to depend on our pathologist and a pathologist must be able to identify the changes not only of benign proliferation but of the type which Dr. Binkley mentioned as atypical changes, which also must be classified as benign, I believe, and not malignant. I would like to ask him to discuss that and see which group he places them in, because we believe that those should be classified as benign and treated as benign lesions. But when malignancy does develop in this particular type of papillary tumor, hidden perhaps somewhere in the base, the most radical treatment is in order.

RUDOLPH V. GORSCH (Flushing, N. Y.): I think that no matter how much experience you have had in proctoscopy or looking at these tumors, it is just impossible for you to tell whether some of these tumors are benign or whether they are malignant. Therefore, I think that one of the most important things to do is to try, as Dr. Marino says, to get the whole tumor. I know that everybody does not agree with that because the snare removal of a tumor, of course, (there is no question about it) is sometimes a little bit difficult and sometimes you are really apt to pinch a part of the bowel wall along with your tumor. But certainly I do not see how you can get anywhere at all unless you get the whole tumor out and examine the whole tumor, because, as has been brought out many times before, the periphery may be malignant or the base can be malignant. If you destroy the whole tumor, of course, you have no evidence at all.

Just a word about the technic of taking these tumors off with a snare. If you want to have a snare where you can push the loop in and out and adjust the snare wire over the top of the tumor, that facilitates the removal very, very easily. If you have a fixed wire there before you try to put it over the tumor, sometimes you are under a considerable amount of difficulty in getting the wire just where you want it on the pedicle. I never put forceps on the tumor at all because I think that is the way you are apt to pull the tumor out and snare part of the bowel wall along with it. The thing to do is to put a piece of gauze above the tumor or three or four pieces of cotton and the tumor will fall right up there and you can pick it out later on.

The next important thing for you is to put your patient in a position so the pedicle of the tumor will hang down. Of course, you have to have proper apparatus; you have to have a proper table and all for that. Then you put your snare in there and you just put the wire around the pedicle and I think an important thing is to put the wire so you can turn the snare and you can actually see the pedicle move on the bowel wall. Then when you snare the tumor off, you have very little difficulty. You will have very little danger of including some of the bowel wall.

After I have done that, I invariably take a suction coagulation handle which I have devised and I (I do not say fulgurate) desiccate the base of the tumor. I desiccate a considerable area around the base of the tumor in order to destroy any lymphatics or possibly any tumor cell which is out around, possibly a centimeter around the tumor.

A distinction should be made in the terminology of the type of current which you are using.

Fulguration is a general term and I do not think proctologists should use it. You either use the Oudin current, the desiccating current or a coagulating current. There is a great deal of difference in the electrosurgical Oudin current and the coagulating current. I think we ought to know which current we are using and we ought to tell our audience which current we are using. Fulgurating is just a general term. With coagulating current you can get into a great deal more trouble because it penetrates the tissues more easily.

There is one other important point about this whole symposium, that is the follow-up. It has been my pleasure recently to present a series of patients treated by Dr. Yeomans many years ago and also to see patients treated by other proctologists around New York City. They come back to me and I get a history on them. In some instances they had had a polyp removed by Dr. Yeomans some twenty or twenty-five years ago. Then, in another case someone had a polyp removed six months ago. I said, "Did the doctor tell you to come back?" "No."

Of course, obviously, part of the treatment is follow-up. If you take a tumor out, you do not know whether it is benign or malignant, no matter how much experience you have. The important thing for you to do is follow up the patients at least periodically every three months.

A. W. MARTIN MARINO: Mr. Chairman, may I have a word?

PRESIDENT RICKETTS: Yes.

DR. MARINO: The hardest decisions I have to make are in those adenomas which the pathologist reports malignancy. Then I go to the pathologist and we go over the situation together. I agree perfectly that a competent pathologist is a most necessary accessory in the performance of these operations. Fortunately, papillary adenomas make a small percentage of all adenomas and the question does not arise too frequently, but I have seen what have been reported as benign adenomas by competent pathologists. I have seen cases develop carcinomas on those sites. It may be that something went wrong.

By the same token, I have seen adenocarcinomas, papillary, small, (they talk of small lesions in this discussion) which have healed perfectly. The mucosa has been so restored that it would be almost impossible to find the

location where that adenocarcinoma had been removed. In following these cases, a carcinoma developed behind. In other words, you can get a fine looking result on the outside; but, even though you have a biopsy, you do not know how much invasion of the various coats has taken place. You are never sure whether or not instead of curing that patient you have simply postponed the eventual diagnosis and perhaps hastened, if not caused, his death.

E. G. MARTIN (Detroit, Mich.): I simply want to warn against fulgurating or desiccating above the rectosigmoid. I do not think the Doctor intended to leave that impression, because you can very readily burn a hole into the peritoneal cavity.

I also would like to say that if we have a sessile tumor in the sigmoid, I believe it should be taken out. I want particularly to express my enjoyment and appreciation of Dr. Swinton's paper and Dr. Laird's discussion.

GEORGE E. BINKLEY (closing): I am not quite sure that I got over what I set out to do. I was not discussing small tumors. I was discussing angiopapillomas, whether they were as big as the end of a pin or whether they were as big as two hands. Moreover, I was not discussing adenomas as we see them; I was discussing a brand, a breed of tumor which we ought to know something about as proctologists, if anybody should; I do not believe we do, I do not and I do not believe you boys do, either.

It is true there are two types of rectal tumors as far as one is malignant and one is benign. I am confining my remarks to the papillary adenoma which I think we ought to recognize and not mix up with everything else. If we do not recognize them, who will? It is possible to do it clinically if you will spend a little time and see enough of these tumors. There is no use saying it cannot be done just because it never has been done; because you cannot do it is not saying someone else cannot do it. We are going to go ahead and you have got to go ahead or stay static or go back; one usually goes back. It is possible clinically to recognize these tumors in a great many cases and they are real problems, as Dr. Scarborough brought out. He has had a little experience and he recognized them for years; he knows what we are talking about.

You take the benign and malignant lesions, yes, and the papillary adenomas, there are benign and malignant lesions but there was

scarcely anybody who recognized them, because I did not tell you much about those atypical changes. That is a pathologic or histologic job, but that is where they fall down. What good is your prognosis if your man or your pathologist does not recognize atypical changes? There are atypical changes, as I see them. Patients come to me with a diagnosis of carcinoma, grade anywhere from 1 to 4, and when I hand them over to Dr. Stewart at Memorial, he says, "There isn't a bit of malignant change in that site. There are atypical changes." It is still a non-malignant polyp. If you are going to take those out and treat them as malignancy, they may become malignant later; but I do not think that man wants a radical resection just because he happens to come and your man or you or somebody else makes a bum diagnosis and he loses his rectum. That is the point I am trying to get over here, that this type of disease has atypical changes and they are still polyps or non-malignant tumors while they have them, until they go on to the malignant group. You have to have a pathologist to recognize that, otherwise you will have 100 per cent five-year survivals no matter what you do with them.

I am not criticizing anybody. I am trying to get you gentlemen to see what I am driving at. Maybe I am all wrong; if I am, why then that is too bad. That is the point I am trying to get over. These things can be classified and the most mistakes are made by pathologists recognizing atypical changes and calling them some form of malignancy, 1, 2, 3 or 4. I can show you patients who have come with the slides and the diagnosis and I have made some enemies among my colleagues because I would not do a radical resection on a patient who had atypical change. They thought I was trying to get the patient away by telling him he did not need an operation.

Someone once took a rectum out of a patient with benign or atypical changes. Even some of the people who have the label of cancer on them have not even got atypical change. What most of you want is a good pathologist. I do not know what I would do without the one we have.

Another point is that Dr. Laird did not think much about radiation therapy. Well, this involves an old age group of patients as a rule; some of them are eighty-five, some are seventy-five, some seventy-eight. Do not think that

you are going to cure all of those people with large papillary adenomas, some of them with atypical changes, some of them with carcinoma; but you can control them until they go out with a cardiac or some other complaint. If you are setting out to do radical resections in all the papillary adenomas, maybe you will get away with it; but I will turn some of mine over if you want to do radical sections. I do not believe I can do it. In that group of cases you can use your radiation to a good degree. You may not cure them, they may be symptom-free for a while and you may have to go back to your fulguration.

I am not going to be pulled into a discussion of treatment. That is too big a job for one day. This is a problem, I think, we should give a good deal of consideration to and it might be wise sometime to write it up and get two or three good pathologists to teach us something about it, because as clinicians we do not know any too much. We can certainly stand some straightening out and some good slides and it might be a benefit not only to ourselves, but also to the pathologists in our own hospitals.

NEIL W. SWINTON (closing): I appreciated Dr. Binkley's paper very much and I was particularly interested in it because I know exactly, I think, what he is talking about. There is very little in the literature. I see among the polyps we find in the clinic a large number of the type that he has described, which I have recognized as being a different entity in many ways from the true adenoma.

I would like to ask you one question. I have not been too impressed with the malignant potentialities of those tumors and yet I have had quite a number that have recurred locally over a period of years. I have had several of those patients whom I have followed between five and ten years. They come in and see me about every six months or once a year, and I find a local recurrence. I burn it off and they are all right for another year. I would like to ask him if he has had a similar experience.

Two or three other points: The use of radium and x-ray, I think, is a personal matter. Our department of radiology does not approve of its use in gastrointestinal malignancy and we have had no experience with it.

The case Dr. Laird mentioned with the early metastasis, I am certain, is going to happen to

us one of these days if we do many of these, and I think it must always be considered.

I wanted to say a word about x-ray in the diagnosis of polyps in general; I did not do it in the previous discussion and it was not brought out. We have been very unhappy in our department of radiology in the number of tumors that we are picking up at an early stage. We know we are not finding a great number of them. A lot was said about contrast air enemas and I believe that you must realize that you are going to miss certain tumors both ways. With proper and complete colon investigation, I believe very strongly they should have both a barium enema and a double contrast. We do them at different times and with a complete repetition of preparation of the colon for each examination.

Another thing, if we find a polyp or polyps, we have learned that we still cannot depend entirely on our radiologist and we will wait three or four weeks and repeat the double contrast air enema; if the polyp is in the same place, then we operate. Many times the polyp has moved.

I wanted to say one other thing, in general on this whole polyp question. Last year in the United States there were 296,000 people who died of malignancy. The last figures I saw on a breakdown for cancer of the colon and rectum were for 1943, at which time there were around 8,000 people dying of cancer of the rectum and colon. We have demonstrated there is a premalignant lesion. What percentage of all the cancer it is, I do not know. I personally think it is at least half. I think that the members of this group, representing certainly in this country the men most interested in this field, have a mission. I think that if we could have everybody in the United States over thirty-five years of age proctoscoped, you would be simply astounded at what we would find.

We are conducting at the clinic on a small scale a sigmoidoscopic examination as a part of a routine physical examination in older patients. We are not able to do it on a wholesale basis. Those figures I will have in another year or two, but we are finding premalignant lesions in patients over forty or fifty years of age in at least 7 per cent of all the patients who go through our office. I know we are saving many patients from cancer.

I enjoyed the discussion very much, and I wish to apologize to Dr. Marino and Dr. Laird in that my paper was negligently written very late and I was not able to send a copy to them in advance.
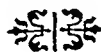
EXTRARECTAL AND EXTRASIGMOIDAL MASSES*

PROCTOSIGMOIDOSCOPIC INTERPRETATION AND EVALUATION

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MOST or many of us who practice in the field of proctology are engaged in the teaching of proctologic fundamentals to undergraduate and graduate students. It is a gratifying experience and while time-consuming is not without its rewards. For a number of years I have stated among other dogmatic assertions that no specialty in medicine can offer a higher percentage of accurate diagnoses than proctology. As we know all too well, the patient's history is often inaccurate and misleading. Thus, the carcinoma patient denies bleeding; constipation to one patient is diarrhea to another. The history rarely differentiates the disease but it should always direct attention to the need for a systematic, careful study of the anus, rectum and colon. With instruments of precision including an experienced index finger, the sigmoidoscope and the roentgen ray, the margin of error should be slim indeed. This is what we teach our students; it is of this that we talk when addressing county and other local medical groups.

During recent years it has become more and more apparent to me that there are certain intangibles in proctology. Most of us have been content with the common phrase "extrarectal or extrasigmoidal mass." Following our examination, which does not demonstrate but makes obvious the presence of such a mass, we submit a written report describing the location of such an impinging mass, its degree of fixation, its consistency, the presence or absence of tenderness and other rather nebulous conclusions. Rarely do we offer a suggestion as to its exact nature. This is probably a correct attitude because usually we do not know.

In reviewing my own records over a twelve-year-period I found to my great surprise that the diagnosis of extrarectal or extrasigmoidal mass or swelling had been entered into the cross files on 313 different patients. Similarly, I was delighted to find that an absolute diagnosis was ultimately established in 284 of these persons. It is thus possible to analyze such a group of case histories and to check the proctosigmoidoscopic findings and the tentative impression gained against the final definitive diagnosis.

A characteristic case history will outline the problem as it often presents itself:

CASE REPORT

Mrs. L. L., aged thirty-nine years, was referred to me in May, 1946 and brought to my office by her brother who is a physician. Briefly, her history of three months' duration was one of short episodes of diarrhea lasting a day or two. There was cramping in the lower abdomen. Twelve days prior to my seeing her, there was a sudden desire to defecate and she passed a small amount of bright red blood. She mentioned a sense of rectal pressure. There had been no weight loss. The cramps and diarrhea were not related to the menstrual periods. There were no other complaints. Proctosigmoidoscopic examination was attempted and I quote from my report. "On digital examination there is a very hard nodular mass felt in the cul-de-sac. It feels extra rectal. Sigmoidoscope passed 11 cm. At this point the mucosa is edematous and thrown into folds. The bowel is sharply angulated and the mass appears to be anterior to the upper rectum. No type of sigmoidoscope could be passed beyond this level. No blood streaks were seen. No intrinsic rectal lesion noted other than small internal hemorrhoids." It was my impression that the palpated lesion was malignant but its

exact nature and origin was in doubt. Roentgenographic examination of the colon was attempted and the report was that there was a point of blockage of the upper rectum beyond which barium would not pass. (In passing it might be mentioned that the roentgenologist thought it indeed strange that I could not visualize such a low lying lesion.)

Surgical and gynecologic consultants were called upon and the final conclusion was that a pelvic malignancy existed and that exploratory laparotomy was justified. As a result of such pooling of our meagre knowledge, the patient was thoroughly prepared preoperatively so that surgery of any magnitude could be done if necessary. At operation a carcinoma of the mid-sigmoid was found which had become fixed to the lower part of the uterine fundus. Combined abdominoperineal resection including hysterectomy was done and to date the patient is quite well.

This case illustrates and perhaps exaggerates some of the difficulties encountered. In most instances the problem is much easier.

As I began to assemble these data a thorough review of all previously published material seemed to indicate that to the best of my knowledge only one prior effort has been made to classify the various extra-rectal and extrasigmoidal masses and to determine their causation. This was a paper published in the Journal of the American Medical Association of July 19, 1944 by Drs. Buie, Jackman and Vickers. It was an excellent and concise presentation based upon 254 patients with malignant tumors of the rectovesical and recto-uterine spaces as had been noted by the examining proctologists of the Mayo Clinic.

An extrarectal mass may feel malignant but prove inflammatory and an occasional extra lower bowel mass may on palpation suggest inflammatory tissue and prove to be malignant. It seemed advisable, therefore, in this review to include all extrarectal and extrasigmoidal masses when on palpation or on proctosigmoidoscopic examination the exact nature of the mass could not be determined with accuracy.

What brings such a patient to the proc-tologist? Curiously, the symptoms were referable to the lower colon or rectum in 61 per cent of the total patients. In many instances the mass was felt by the patient's own physician and to his credit he had believed that proctoscopic examination was indicated. A few were referred for proctoscopic examination as a preliminary to roentgenographic studies of the gastrointestinal tract.

Analysis of the total (313) patients with extrarectal and extrasigmoidal masses revealed the following:

TABLE 1
TYPE AND ORIGIN PROVEN

Method of Proof:	
1. Biopsy	6
2. Roentgenograms	77
3. Gynecologic study	57
4. Urologic study	20
5. Laparotomy	51
6. Rectal operation	55
7. Autopsy	18
Total Cases with confirmed diagnosis	284
Total Cases with unconfirmed diagnosis	29

In twenty-two instances the failure of confirmation of diagnosis was refusal on the part of the patient to submit to further investigation. In seven fatal cases autopsy was not obtained.

It will be noted that biopsy as a means of obtaining microscopic proof of the diagnosis was rarely employed. It is with the greatest hesitancy that I offer to puncture an intact mucosa to obtain a microscopic section. Certainly I would never do so if I knew the mass was above the peritoneal reflection. Other and safer means exist to confirm one's suspicions. Since the term "rectal operation" in the above table would appear to be merely an extensive biopsy, I would hasten to add that in the series studied I have, perhaps unwisely, included all perirectal abscesses at or above the level of the levator ani muscles.

A detailed discussion of each of the above listed diseases is neither practical nor possible. However, a few general statements seem advisable: When the finger or the end of the sigmoidoscope encounters resistance and pressure causes intense pain, we are in

all likelihood dealing with an inflammatory rather than a neoplastic entity. Such subjective evidence is by no means absolute but it is highly suggestive. The duration of symptoms, if any, must be carefully analyzed. Thus, the patient who complains of

TABLE II

CONFIRMED DIAGNOSIS AND ORIGIN OF LESION IN 284 PATIENTS

Diagnosis	Total Cases
I. Perirectal or pelvic abscess—levator level or higher	47
II. Presacral cysts and dermoids	4
III. Pelvic disease (female)	77
(a) Leiomyomatous uterii and displacement	21
(b) Tubo-ovarian inflammation or abscess	29
(c) Endometriosis	17
(1) Pelvic organs	9
(2) Rectovaginal septum	5
(3) Sigmoid	3
(d) Ovarian cystic disease	4
(e) Carcinoma uterus, ovaries	5
(f) Carcinoma bladder	1
IV. Genitourinary tract (male)	20
(a) Seminal vesiculitis	2
(b) Carcinoma prostate	15
(c) Carcinoma bladder	2
(d) Prostatic abscess	1
V. Diverticulitis.	74
VI. Rectal shelf implants (Blumer)	23
VII. Retroperitoneal sarcoma, carcinomatosis	11
VIII. Perirectal tumors	8
(a) Tumors of ischio anal fossa	5
(b) Perirectal carcinoid	3
(c) Squamous epithelioma in deep fistula tract	1
IX. Carcinoma sigmoid	20

pain deep in the pelvis of only a few days or weeks duration is again likely to be found harboring an inflammatory process. This might be a smouldering pelvirectal abscess, gynecologic or urinary tract infection and of course subacute, acute or ruptured diverticula of the sigmoid.

A hard, fixed, nodular, non-tender mass palpated through the anterior wall of the mid-rectum will rarely prove to be anything except carcinoma or malignant implants. The original lesion must then be sought. In this review the stomach seemed to be the primary focus in more than one-half of the patients.

It is known that an ulcerated inflamed diverticulum of the sigmoid will produce not only the symptoms of constipation or diarrhea or both, with increased gas and

left lower abdominal pain, but may produce bleeding as well. It is my belief, however, that bleeding caused by diverticulitis is comparatively rare and to me this presence or absence of bleeding is the outstanding differentiating feature between sigmoidal carcinoma and diverticulitis.

Lesions of the genitourinary tract in the female which are producing low colonic symptoms are rarely of short duration. The past history is often illuminating. Periodicity of symptoms, especially if related to the menstrual periods is significant. Such a relationship in women with endometriosis is well known.

Reexamination of Table II should quickly convince the reader that establishing the ultimate correct diagnosis is rarely a "one man job." Help is needed and occasionally multiple consultations are required. The proctologist may speculate as to the underlying pathologic lesion but his conjectures should not cause undue delay.

If a shadow of a doubt exists as to the actual diagnosis (and such doubt will exist more times than not), then let him call upon his colleagues the roentgenologist, the gynecologist or the urologist. By such means ill advised or hasty laparotomy performed upon a poorly prepared patient will usually be avoided.

In mentioning the paucity of previously written reports, I would hasten to add that many splendid studies have been published each dealing with one of the single manifestations of extrabowel lesions. Thus, among others are the papers by Blumer concerning the rectal shelf implants; Masson, Cullen and many others on perirectal and perisigmoidal findings in endometriosis. Especially would I recall to your attention the excellent presentation by Jackman in 1942 on the proctosigmoidoscopic findings in diverticular disease of the sigmoid.

COMMENT

From this brief discussion of some of the features in diseased conditions producing extrarectal and extrasigmoidal masses it

can be seen that in many instances certain although perhaps vague characteristics are present. If critically analyzed, an attempt at differential diagnosis is justifiable. Certainly such an attempt is an interesting speculation.

One may properly ask: "Why should the proctologist worry as to the pathological nature of a lesion which he cannot see?" My answer, if one is needed, is that the proctologist in every possible way must lend his help and knowledge to the referring physician and for the benefit of the patient.

The proctologist need not act as gynecologic, urologic or roentgenologic consultant. Others render such service but he should as a result of his examination be able to sit down with the patient and the referring physician to discuss the possibilities, the method of approach, the need for further consultation and perhaps touch on prognosis. Such a consulting proctologist will then have rendered a far greater service than merely to have written as his report "Extrarectal or extrasigmoidal mass: type unknown."

A CARBUNCLE is simply an extensive connected furunculosis. The Staphylococcus is the most frequently found organism. This lesion is quite uncommon in the perianal skin.

The brief excerpts in this issue have been taken from "Ambulatory Proctology" by Alfred J. Cantor (Paul B. Hoeber, Inc.)

PRIMARY RESECTION OF THE COLON AND RECTUM WITH PARTICULAR REFERENCE TO CANCER AND ULCERATIVE COLITIS*

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THE surgeon's primary concern with the problem of visceral malignancy up until quite recently has been with operative mortality. Perusal of the literature confirms this suggestion. The improvement which has come about in surgery generally within the last few years definitely suggests that the surgeon, now having improved considerably his record with reference to operative mortality, has begun to focus his attention more earnestly upon the problem of ultimate cure of cancer.

The perineal operation for rectal cancer and posterior excision of the rectum preceded by colostomy are reserved largely for the occasional patient for whom the conventional, abdominoperineal operation does not seem well suited. In most clinics professing an interest in the problem of rectal cancer the abdominoperineal operation has become the procedure of choice. In this clinic during the past five years, attempts have been made to ascertain whether an abdominal dissection directed at removal of the lesion and the lymph node drainage area, accompanied by reestablishment of intestinal continuity, is a satisfactory operation for rectal and rectosigmoidal cancers.

That such an operation can be done with an operative mortality not out of line with that of the abdominoperineal operation is apparent in our own experience; that the operation is followed by satisfactory continence in most patients, especially when the end-to-end suture method has been employed, also has been established; that this more conservative operation cannot have as high an ultimate cure rate for rectal cancer as the abdominoperineal operation is also apparent in that omission of the perineal portion of the operation, particularly salvage of the levators, constitutes a less radical operation for rectal cancer.

From our own experience it is yet too early to give an opinion in the matter of how much less efficient the more conservative operation is than the standard abdominoperineal operation. That is the particular point still to be clarified. Already in 1944, after two years' experience with the operation in which sphincteric function was preserved, it was apparent that the operation was unsuited for dealing with low lying lesions in juxtaposition to the levators,[14] as well as for large, bulky, fixed cancers presenting lymph node involvement.

Accumulated experience from a number of sources suggests definitely that no surgeon employing even the most radical operation can lay claim to any important achievement in dealing with rectal cancers exhibiting lymph node involvement which fall into Dukes' Group C Classification.[3,4] Earlier detection and earlier operation for rectal cancer are without question the items which must be looked to, in order that a more hopeful prospect will come about in the field of rectal cancer.

CANCER OF THE COLON

In cancer of the colon it is possible to do a one-stage excision with removal of the

lymphatic drainage area, preserving intestinal continuity in all operable cases. The Bloch-Paul-Mikulicz operation is an incomplete operation for colic cancer, and should be discarded by all experienced visceral surgeons. No surgeon should be found advocating general adoption of the abdominoperineal operation for rectal cancer, at the same time employing the exteriorization operation for cancer of the colon. Such practice smacks of inconsistency and suggests that the surgeon's mastery of the abdominoperineal operation has superseded his accomplishment in the one-stage end-to-end anastomosis for colic cancer.

Our associate, Dr. David State,[10] has advocated an extension of the abdominoperineal operation for those patients who exhibit lymph node involvement in the upper end of the inferior mesenteric artery pedicle; in such patients, he excises the entire left colon, together with the mesentery, establishing a colostomy in the transverse colon. The latitude for this extension of removal of the lymphatic drainage area is even wider in anastomotic operations. When numerous and enlarged lymph nodes are found in juxtaposition to a colic lesion, it has been routine practice for some years in this clinic to remove a considerably larger segment of the colon, thus enlarging the opportunity of getting wide of the lesion. Especially in lesions of the left colon is such a practice desirable. Having observed instances of recurrence about the ureter and the aorta after incomplete excision of the lymphatic drainage area in cancers of the descending colon, excision of that segment together with the splenic and sigmoid flexures, establishing continuity by anastomosing the transverse to the terminal pelvic colon has become quite routine practice in this clinic for such lesions. Moreover, in a few instances, because of the finding of enlarged lymph nodes in the mesentery of even more proximal reaches of the colon, the greater portion of the colon has been excised, anastomosing the ileum to the terminal

pelvic colon. In some instances the lymph nodes have been reported by the pathologist as benign. Yet, this circumstance is less embarrassing to both patient and surgeon than is incomplete removal of the lymph nodes involved by the cancer process. Surgeons must now and then have occasion to entertain some misgivings over such negative reports of lymph node involvement. Obviously the difficulty of recognizing a few cancer cells in a lymph node must be great. The biologic test constituted by the elapse of time, to be certain, is a sure test to determine whether the pathologist's observation was correct but it is not a risk to be run with the interests of the patient in mind. Excision of an increased length of colon does not change the character of the surgical problem particularly and if one were to adopt this policy uniformly, the surgeon would undoubtedly be rewarded sufficiently often by the finding of unsuspected polyps as well as an occasional additional undetected cancer to justify this extension of effort.

In malignancies of the right colon the sentinel node at the inferior border of the pancreas alongside the mesenteric vessels is to be sought out and removed. In cancers of the transverse colon, the splenic and hepatic flexures should be unhinged from their phrenocolic attachments, thus permitting removal of a wider segment of the mesentery, insuring at the same time adequate mobility of bowel to effect an end-to-end anastomosis without tension. Among the patients included in this study are three patients in whom it was necessary to anastomose the proximal third of the transverse colon to the rectum because of extensive lymph node involvement in the inferior mesenteric artery lymphatic pedicle. Inasmuch as sacrifice of a good portion of the sigmoid colon voids the possibility of restoration of continuity between rectum and descending colon, the only alternative is mobilization of the transverse colon for the anastomosis. If the marginal vessels are well developed, the mid-colic artery may even be divided near

its origin and when the hepatic flexure and ascending colon are freed up, there is ample length of gut for an anastomosis to the mid-rectum.

To those surgeons who are committed to employment of the open anastomosis, the antibiotics such as the sulfonamides employed as intestinal antiseptics or streptomycin are a great boon. The experience of this clinic with primary resection, employing the closed anastomosis in the pre-intestinal antiseptic era (1941 to 1943) strongly suggests that employment of antibiotics is not a *sine qua non* of a successful operation.[12] In that two year interval sixty-one consecutive colic resections were done including the rectosigmoid area with one death, a hospital mortality of 1.6 per cent which is an accomplishment we have been unable to duplicate since, even with preoperative administration of intestinal antibiotics to occasional patients.

In every operative series of some size, when one is dealing with cancer, there enters always the item of unavoidable deaths; that is, losses in the postoperative phase from coronary and cerebral artery thrombosis and similar causes. The time may come when even death from pulmonary embolism will be looked upon as an avoidable cause of death. In the 1941 to 1943 experience there were no unavoidable deaths; in the 1943 to 1945 period, during which seventy-eight colic resections were done, there were six hospital deaths, a mortality of 7.6 per cent; three of the deaths, however, were owing to unavoidable cause. In other words, in the first series we used up a lot of surgical luck in having no unavoidable deaths. The score was evened up by the law of averages in the second series, giving us an overall hospital mortality of 5 per cent for both series.[13]

THE PRESENT STUDY

It is the writers' intent in this effort to indicate what the experience of this clinic has been with anastomotic operations for rectal and rectosigmoidal lesions. It is still too early to assess fully the accomplishment of the low anastomosis in dealing with primary rectal malignancies. Yet, enough experience has been accumulated to suggest what the outlook may be for the dual objective of curing the cancer and, at the same time, salvaging sphincteric function.

A serious interest in the problem of the low anastomosis with preservation of sphincteric function in this clinic dates back to 1942. It is the experience of those years which will be reviewed herein. Essential data concerning the patients and the operations are to be found in the tables. We have divided somewhat arbitrarily the surgical problem into two categories: (1) Those lesions situated within 13 cm. from the anus are regarded as being present in the true rectum; (2) those lesions beyond 13 cm. and not more than 20 cm. from the anus are considered as being in the rectosigmoid area. The term rectosigmoid is also a rather arbitrary and not strictly an anatomic designation. However, prior to the BNA reclassification of the constituent parts of the pelvic colon and rectum, the colon pelvinum, that segment of the colon between the distal end of the sigmoid flexure and the present arbitrary upper limits of the rectum (the third sacral vertebra) was included in the rectum. Surgeons, generally, employ the appellation rectosigmoid to this segment of colon.

In others words, in this study the operations have been divided into a high and low group. No lesions situated beyond 20 cm. from the anus have been included in this study. The majority of the patients had primary carcinomatous lesions of either the rectum or the rectosigmoid. In addition, there is a miscellaneous group constituted by a variety of conditions including primary lesions of the rectum which on microscopic study proved to be benign, extrinsic tumors involving the rectum and a few instances of diverticulitis. There is also a third group constituted by patients with ulcerative colitis for whom primary intestinal resection and anastomosis was done.

In this latter group, inasmuch as it was considered desirable to include all the patients having primary resection for ulcerative colitis, there are one or two included in whom the lower level of the lesion was a little higher than the arbitrary 20 cm level from the anus.

OPERATIVE TECHNIC EMPLOYED

The majority of the patients were operated upon employing the end-to-end suture technic. In the main, it can be said that one can make a satisfactory anastomosis at a level lower than it is safe to cure cancer. Because of the depth of the wound, a single row of sutures must suffice for the anastomosis. Interestingly enough, the results have been so satisfactory that we have come to employ a single row of interrupted silk sutures (0000) as the standard manner in which to effect an anastomosis anywhere in the gastrointestinal canal. We employ the Lembert type of stitch, spacing the sutures approximately 3 mm. apart. This somewhat close placement of the stitches makes for a rather large number of sutures, approximately forty sutures in the ordinary anastomosis. A large and patulous stoma follows this scheme of anastomosis quite uniformly.

The low anastomosis has been made by suture from within the abdomen as low as 3.5 cm. from the anal orifice with primary healing without formation of a sinus or fistula. The number of instances in which an anastomosis has been effected at as low a level as 5 cm. from the anus without temporary fistula formation, however, is few. Moreover, inasmuch as our own experience suggests quite definitely that even in the early lesion the very low anastomosis is contraindicated because of the likelihood of incomplete removal, the necessity for performing anastomosis at levels below 5 cm. is not frequent. The abdomino-anal pull-through operation has a limited indication. Inasmuch as healing is slow with this method, we have come to employ complemental colostomy in all such instances. Complete fecal deviation can be

achieved by a single loop colostomy.[14] Then, when the segment of the pelvic colon pulled through the anus has healed securely, the colostomy can be closed. Healing in the direct suture anastomosis is ordinarily rapid, permitting dismissal of patients from the hospital usually within a week of performance of the operation. In patients in the upper group of rectal lesions (14 to 20 cm.) dismissal from the hospital five to six days after operation is the rule, just as in colic resection in the more proximal reaches of the colon barring complication. The operation without complication is the surgeon's first objective. It is synonymous with low mortality as well as low morbidity. Details of the technic in the performance of these operations have been described elsewhere and will not be repeated here.

CASE GROUPING

From Table 1, it is to be noted that during the years 1945 and 1946 at the University Hospitals 78 per cent of patients admitted to the surgical wards for treatment of cancer of the rectum or rectosigmoid underwent some type of radical operative procedure. In 56.4 per cent of instances the abdominoperineal operation was done and in 43.6 per cent the more conservative procedure of excision of the cancer with sphincter preservation was done.

Among the eighty-seven excisions of the rectum or rectosigmoid with sphincter preservation reported herein, there were sixty-three (72.4 per cent) so-called curative operations; the remaining twenty-four (27.6 per cent) were palliative excisions with restoration of bowel continuity. In this study the following criteria have been employed to characterize the palliative resection: (1) hepatic metastases; (2) peri-aortic lymph nodes or evidences of peritoneal metastases other than in the inferior mesenteric lymphatic pedicle; and (3) indurated lesions presenting high grade fixation to adjacent structures. The majority of these palliative resections fell

into the first category, i.e., patients with demonstrable hepatic metastases.

Table I suggests, in the main, that the pathologic grading or division of cases essentially employing Dukes' classification is that essentially reported in larger series

TABLE I

RESECTABILITY RATE FOR THE YEARS OF 1945 AND 1946 FOR PRIMARY CARCINOMA OF THE RECTUM AND LOW PELVIC COLON

	No. Cases	Resectable	Nonresectable
Jan. 1, 1945 to Dec. 31, 1945...	41	34	7
Jan. 1, 1946 to Dec. 31, 1946...	59	44	15
Total Jan. 1, 1945 to Dec. 31, 1946..................	100	78	22

	No. Cases	Per Cent of Resectable Cases
Abdominoperineal operations... (Both years)	44	56.4
Anastomotic operations........	34	43.6

in which the abdominoperineal operation has been carried out. In a study of the St. Marks Hospital material, Gabriel[5] reported the following division of their cases: Dukes' group A, 15 per cent; group B, 36 per cent and group C, 49 per cent. In this present series of anastomotic procedures, resection was undertaken as a palliative operation in twenty-four patients or 27.6 per cent of the eighty-seven patients in the group. Of the eighty-seven patients subjected to operation (Table I) 52 per cent fell into Dukes' groups A and B and 48 per cent into Dukes' group C. However, the division between the A and B cases was somewhat different in the present series, 26 per cent in each group, as contrasted with 15 per cent for Group A and 35 per cent for Group B as reported in the study by Gabriel in 1936.

However, amongst the sixty-three curative operations undertaken in this series,

the division of patients was as follows: Dukes' group A, twenty-three patients (36.5 per cent); group B, eleven patients (27 per cent) and in group C, twenty-three patients (36.5 per cent). In other words, the case alignment in the curative group of

TABLE II

CANCER OF THE RECTUM AND LOW PELVIC COLON RESECTION WITH PRESERVATION OF THE SPHINCTERS January, 1942 to June, 1947 Pathologic Classification of All Cases

Dukes' Group	No. Cases	Per Cent
A	23	26
B	23	26
C	41	48
	87	100

Pathologic Classification Curative Cases

Dukes' Group	No. Cases	Per Cent
A	23	36.5
B	17	27.0
C	23	36.5
	63	100.0

operations suggests definitely some case selection.

SUMMARY OF RESULTS

It is obviously too early to attempt to assess in a final manner the results of treatment of the more conservative operation in the cure of cancer of the rectum and rectosigmoid. Our experience with the procedures is summarized in Tables II to XI inclusive. This analysis is recorded in some detail in the hope that a study of the tables may prove helpful to others in assessing the worth of anastomotic operations in dealing with cancer of the rectum. Briefly summarized, these data suggest: (1) Restitution of intestinal continuity may be reestablished as a one-stage operative procedure after excision of the terminal pelvic colon and upper rectum with a hospital mortality that is in keeping with

that attending performance of the abdominoperineal operation. (Tables III and IV.)

TABLE III

DIVISION OF CASES AND OPERATIVE MORTALITY RESECTION WITH PRESERVATION OF THE SPHINCTERS FOR ALL LESIONS OF THE TRUE RECTUM AND LOW PELVIC COLON

January, 1942 to June, 1947

Procedure and Indication	No. Cases	No. Deaths	Per Cent Mortality
A. Resection and Primary Anastomosis			
1. Primary cancer of the true rectum (0–13 cm.)	51	3	5.88
2. Primary cancer of low pelvic colon (14–20 cm.)	20	1	5.00
3. Secondary malignant invasion			
a. Carcinoma of the ovary	2	0	0.00
b. Carcinoma of the cecum	2	0	0.00
4. Benign polyps	5	0	0.00
5. Diverticulitis	4	0	0.00
6. Benign post-irradiative stricture	1	0	0.00
7. Endometriosis	2	0	0.00
8. Non-specific ulcer of the rectum	1	0	0.00
9. Chronic ulcerative colitis	11	0	0.00
10. Tuberculous stricture of the rectum	1	0	0.00
11. Restoration of continuity after colostomy and inversion of rectal stump	1	0	0.00
Total	101	4	3.96
B. Resection and Pull-through of the Colon			
1. Primary cancer of the rectum (0–13 cm.)	16	1	6.25
2. Retroperitoneal lymphoblastoma	1	0	0.00
3. Perirectal abscess and fistula	1	0	0.00
Total	18	1	5.55
C. Resection and Pull-through of Ileum for Chronic Ulcerative Colitis	2	0	0.00
Total All Cases	121	5	4.13

(2) Functional sphincteric control is *complete* after the suture operation.

(3) Accompanying the abdomino-anal pull-through procedure good sphincteric control is the rule although 100 per cent recovery of function is not consistently

TABLE IV

CARCINOMA OF THE TRUE RECTUM AND LOW PELVIC COLON HOSPITAL MORTALITY IN PRIMARY RESECTION AND PRESERVATION OF THE SPHINCTERS

Type of Procedure	No. Cases	No. Died	Per Cent Mortality
A. Carcinoma of the true rectum (0–13 cm.)			
1. Resection and primary anastomosis	51	3	5.88
2. Resection and Hochenegg pull-through	15	1	6.65
3. Babcock-Bacon procedure	1	0	0.00
Total	67	4	5.97
B. Carcinoma of the low pelvic colon (14–20 cm.)	20	1	5.00
Total	20	1	5.00
Total All Cases	87	5	3.76
Causes of hospital mortality			
1. Pulmonary embolus	2		
2. Coronary thrombosis	2		
3. Uremia	1		
Total	5		

obtained; moreover, the healing phase is longer and it is wise to make a complemental diversionary colostomy to afford rest to the pelvic colon pulled through the remaining distal sphincteric segment. Maintenance of normal continence in the suture operations is owing to preservation of the internal sphincter. Even in suture anastomosis made at 4 or 5 cm. from the anus, continence in these patients has been invariably good. Any operation[1] which eliminates the internal sphincter excludes the possibility of satisfactory continence. Even in the pull-through operation as done in this clinic, in which the rectal mucosa near the anorectal line is removed, (Whitehead maneuver), some injury to the

TABLE V

CARCINOMA OF THE RECTUM AND LOW PELVIC COLON RESECTION WITH PRIMARY ANASTOMOSIS AND RESECTION (BOTH GROUPS) WITH ABDOMINO-ANAL PULL-THROUGH

January, 1942 to June, 1947

No. cases.. 87
Hospital mortality.. 5
No. follow-up... 0

Year	No. Cases	Palliative	Curative	Present Status of the Curative Cases							
				Living		Exp. Ca.[1]		Exp. Other[2]		Loc. Rec.[3]	
				No.	Per Cent	No.	Per Cent	No.	Per Cent	No.	Per Cent
1942	13	3	10	6	60.0	1	10.0	3	30.0	1	10.0
1943	13	2	11	8	72.7	2	18.2	1	9.10	1	9.10
1944	18	8	10	5	50.0	4	40.0	1	10.0	2	20.0
1945	14	2	12	8	66.7	4	33.3	0	0.00	2	16.6
1946	19	3	16	13	81.2	1	6.30	2	12.4	1	6.30
1947	5	1	4	4	100.0	0	0.00	0	0.00	0	0.00
Total	82	19 (23.2%)	63 (76.8%)	44	70.0	12	19.0	7	11.0	7	11.0

[1] Expired carcinoma—expired of the primary carcinoma of the rectum, either recurrence or metastases.
[2] Expired other—expired of causes other than the primary carcinoma of the rectum.
[3] Local recurrence—local recurrence of the carcinoma in the area of the original lesion.

TABLE VI

CARCINOMA OF THE RECTUM AND LOW PELVIC COLON RESECTION WITH PRIMARY ANASTOMOSIS FOR LESIONS 13 TO 20 CM. (UPPER GROUP)

January, 1942 to June, 1947

Total cases... 20
Hospital mortality.. 1
No follow-up... 0

Year	No. Cases	Palliative	Curative	Present Status of the Curative Cases							
				Living		Exp. Ca.[1]		Exp. Other[2]		Loc. Rec.[3]	
				No.	Per Cent	No.	Per Cent	No.	Per Cent	No.	Per Cent
1942	3	1	2	1	50.0	0	0.00	1	50.0	0	0.00
1943	3	1	2	1	50.0	0	0.00	1	50.0	0	0.00
1944	4	3	1	1	100.0	0	0.00	0	0.0	0	0.00
1945	4	1	3	2	66.7	1	33.3	0	0.00	0	0.00
1946	4	1	3	2	66.7	0	0.00	1	33.3	0	0.00
1947	1	0	1	1	100.0	0	0.00	0	0.00	0	0.00
Total	19	7 (36.8%)	12 (63.2%)	8	66.7	1	8.30	3	25.0	0	0.00

[1] Expired carcinoma—expired of the primary carcinoma of the rectum, either recurrence or metastases.
[2] Expired other—expired of causes other than the primary carcinoma of he rectum.
[3] Local recurrence—local recurrence of the carcinoma in the area of the toriginal lesion.

TABLE VII

CARCINOMA OF THE RECTUM AND LOW PELVIC COLON RESECTION WITH PRIMARY ANASTOMOSIS AND RESECTION WITH ABDOMINO-ANAL PULL-THROUGH (0 TO 13 CM.) (LOWER GROUP)
January, 1942 to June, 1947

No. cases........ 67
Hospital mortality........ 4
No follow-up........ 0

Year	No. Cases	Palliative	Curative	Present Status of the Curative Cases							
				Living		Exp. Ca.[1]		Exp. Other[2]		Loc. Rec.[3]	
				No.	Per Cent	No.	Per Cent	No.	Per Cent	No.	Per Cent
1942	10	2	8	5	62.5	1	12.5	2	25.0	1	12.5
1943	10	1	9	7	78	2	22.2	0	....	1	11.1
1944	14	5	9	4	44.4	4	44.4	1	11.1	2	22.2
1945	10	1	9	6	66.7	3	33.3	0	0	2	22.2
1946	15	2	13	11	84.6	1	7.7	1	7.7	1	7.7
1947	4	1	3	3	100.0	0	0	0	0	0	0
Total	63	12 (19.0)	51 (81)	36	70.6	11	21.6	4	7.8	7	13.7

[1] Expired carcinoma—expired of the primary carcinoma of the rectum, either recurrence or metastases.
[2] Expired other—expired of causes other than the primary carcinoma of the rectum.
[3] Local recurrence—local recurrence of the carcinoma in the area of the original lesion.

TABLE VIII

CARCINOMA OF THE RECTUM AND LOW PELVIC COLON RESECTION AND HOCHENEGG PULL-THROUGH FOR LESIONS (0 TO 13 CM.)* (LOWER GROUP)
January, 1942 to June, 1947

Total cases........ 16
Hospital mortality........ 1
No follow-up........ 0

Year	No. Cases	Palliative	Curative	Present Status of the Curative Cases							
				Living		Exp. Ca.[1]		Exp. Other[2]		Loc. Rec.[3]	
				No.	Per Cent	No.	Per Cent	No.	Per Cent	No.	Per Cent
1942	4	0	4	2	50.0	1	25.0	1	25.0	1	25.0
1943	5	0	5	4	80.0	1	20.0	0	0.00	1	20.0
1944	1	1	0								
1945	0	0	0								
1946	5	1	4	3	75.0	1	25.0	0	0.00	1	25.0
Total	15	2 (13.3%)	13 (86.7%)	9	69.2	3	23.1	1	7.70	3	23.1

* No abdomino-anal pull-through operations in the upper group (13–20 cm.).
[1] Expired carcinoma—expired of the primary carcinoma of the rectum, either recurrence or metastases.
[2] Expired other—expired of causes other than primary carcinoma of the rectum.
[3] Local recurrence—local recurrence of the carcinoma in the area of the original lesion.

internal sphincter probably occurs, for the continence in this group of patients is by no means as complete as in the suture group. Moreover, as contrasted with the frequent loss of the sex function in males who have undergone abdominoperineal patients approximately the same promise of cure as does the more radical abdomino-perineal operation. For lesions in this area presenting dubious or borderline operability the abdominoperineal operation is the procedure of choice.

TABLE IX

CARCINOMA OF THE RECTUM AND LOW PELVIC COLON RESECTION WITH PRIMARY ANASTOMOSIS FOR LESIONS 0 TO 13 CM. (LOWER GROUP)

January, 1942 to June, 1947

Total cases... 51
Hospital mortality... 3
No follow-up.. 0

Year	No. Cases	Palliative	Curative	Present Status of the Curative Cases							
				Living		Exp. Ca.[1]		Exp. Other[2]		Loc. Rec.[3]	
				No.	Per Cent	No.	Per Cent	No.	Per Cent	No.	Per Cent
1942	6	2	4	3	75.0	0	0.00	1	25.0	0	0.00
1943	5	1	4	3	75.0	1	25.0	0	0.00	0	0.00
1944	13	4	9	4	44.5	4	44.5	1	11.0	2	22.3
1945	10	1	9	6	66.7	3	33.3	0	0.00	2	22.3
1946	10	1	9	8	89.0	0	0.00	1	11.0	0	0.00
1947	4	1	3	3	100.0	0	0.00	0	0.00	0	0.00
Total	48	10 (20.9%)	38 (79.1%)	27	71.0	8	21.0	3	8.00	4	10.5

[1] Expired carcinoma—expired of the primary carcinoma of the rectum, either recurrence or metastases.
[2] Expired other—expired of causes other than the primary carcinoma of the rectum.
[3] Local recurrence—local recurrence of the carcinoma in the area of the original lesion.

excision of the rectum, preservation of that function is the rule in the low anastomosis.

(4) In lesions of the rectosigmoid (upper segment 14 to 20 cm.) local recurrence has not followed the type of operation described herein. (Table VI.)

(5) The incidence of local recurrence following conservative procedures for lesions at 8 cm. or less from the anus is frequent enough to suggest that sphincter saving operations are contraindicated in all low-lying lesions; with abandonment of the conservative operations for low-lying lesions there will be less necessity for performance of the abdomino-anal pull-through operation.

(6) In the middle rectal segment or rectal ampulla above 8 cm. the conservative operation is a satisfactory operation for *suitable* cases and holds out to such

LOCAL RECURRENCE

The incidence of local recurrence in the curative groups is outlined in Table XI. Among these fifty-one patients operated upon, local recurrence has been observed in seven or 14 per cent of instances. Among these instances of local recurrence five or 70 per cent occurred in lesions at 8 cm. or less from the anus; two or 30 per cent occurred in the resections done for lesions between 9 and 13 cm. from the anus. One of these patients had a large colloid cancer which is notably difficult to cure;[7] the other had a large Dukes' group C lesion in which it became necessary to bring down the proximal third of the transverse colon for anastomosis with the mid-rectum because of the presence of large lymph nodes beyond the reaches of the conventional

excision of the inferior mesenteric artery lymphatic pedicle. Among the twenty-four patients upon whom palliative resection was undertaken local recurrence was observed three times; twice in the groups in which the lesions were 6 to 8 cm. from the

Gilchrist who have been keen exponents of the necessity of liberal excision of the lymphatic drainage area in operations for cancer of the rectum, find on a study of their cases of abdominoperineal operation that the incidence of local recurrence

TABLE X

CANCER OF THE RECTUM AND LOW PELVIC COLON RESECTION WITH PRESERVATION OF THE SPHINCTERS
Pathologic Classification and Survival Rate by Years
Curative Group

Year	Duke's Class	No. Cases	Exp. Ca.		Exp. Other		Living		Survival
			No.	Per Cent	No.	Per Cent	No.	Per Cent	
1942	A	4			1	25	3	75	5 yr.
	B	4	1	25	1	25	2	50	5 yr.
	C	2			1	50	1	50	5 yr.
1943	A	4					4	100	4 yr.
	B	4	1	25	1	25	2	40	4 yr.
	C	3	1	33			2	67	4 yr.
1944	A	5	1	20	1	20	3	60	3 yr.
	B	2					2	100	3 yr.
	C	3	3	100			0	0	3 yr.
1945	A	3	1	33			2	67	2 yr.
	B	1	1	100	..	0	0	0	2 yr.
	C	8	2	25			6	75	2 yr.
1946	A	4			2	50	2	50	1 yr.
	B	6	1	17			5	83	1 yr.
	C	6					6	100	1 yr.
1947	A	3					3	100	Less than 1 yr.
	B	0	..	..	..	..			
	C	1					1	100	
Total..	..	63	12	19	7	11	44	70	

anus and once in the group in which that lesion was 9 to 13 cm. This latter patient exhibited all three of the features which were designated above to characterize the palliative operation in this study.

Almost invariably, the local recurrence has not been *primarily* at the site of the anastomosis, but outside of it, extending into the bowel secondarily, a circumstance which suggests that it is the lateral invisible spread of cancer that the more conservative operation fails to deal with adequately. On this score, however, no operation is free from blame. David and

amongst the five-year survivors was 22 per cent.[3] In other words, the abdominoperineal operation, the most radical of available curative operations, is also an incomplete operation for certain cases. That it may be for any lesion that extends beyond the longitudinal muscles of the rectal wall is apparent on careful scrutiny of any anatomic atlas which depicts the compartments of the pelvic fascia.

Local recurrence is an indictment of any operation and is synonymous with inadequate excision. It is to be noted (Table VI) in those instances in which the curative

operation was undertaken for lesions at 14 to 20 cm. from the anus, there were no local recurrences. This circumstance suggests rather definitely that for lesions in the rectosigmoid the conservative operation is probably just as effective for the

TABLE XI

CARCINOMA OF THE RECTUM AND LOW PELVIC COLON RESECTION WITH PRESERVATION OF THE SPHINCTERS

Cases of Local Recurrence in the Curative Group

Distance in Cm. from Anus	No. Cases	No. Cases of Recurrence	Per Cent of Cases	Per Cent of Total Local Recurrence
0–5 cm. incl.	7	2	30	30
6–8 cm. incl.	12	3	25	40
9–13-cm. incl.	32	2	6.3	30
14–20 cm. incl.	12	0	0	0
Total	63	7	14	100

cure of cancer as is the abdominoperineal operation.[15].

The very circumstance that recurrence is observed after the excision of a low-lying Dukes' Group A lesion outside of the site of anastomosis suggests that this or any other classification subdivides cases occasionally beyond what is justified by subsequent developments. In other words, a demonstrated Dukes' Group A lesion occasionally is shown by the elapse of time and growth of the lateral invisible spread to belong actually in Dukes' Group C. A closer correlation between the presence of venous invasion by the tumor and the Dukes' grouping is in order. That such venous invasion is rather frequent as well as ominous has been established.[2,9] Careful study to note the absence or presence of such venous invasion is important to assess thoroughly the possibility of cure in any lesion. Unfortunately, such a study was not made routinely in this group. It is certain that as one enlarges the indications for operation the number of patients with occult metastases will be larger.

UNSUSPECTED POLYPS IN A MORE PROXIMAL SEGMENT OF THE COLON

The frequency with which unsuspected polyps were encountered in this series is startling and suggests that if one were determined to leave no stone unturned to leave such precursors of colic cancer behind, he probably would be fully justified in excising the entire left colon, anastomosing the proximal third of the transverse colon to the rectum, a feasible operative procedure and one which was performed three times in this series of cases. As was stated previously for lesions in the descending colon, it has become regular practice in this clinic to excise the splenic and sigmoid flexures, anastomosing the transverse colon to the terminal pelvic colon. Certainly when one does the abdominoperineal operation for cancer in the lower and mid-rectal segments it is wise always to remove from above early in the operative procedure that segment of the sigmoid colon beyond the site chosen for colostomy down to the peritoneal reflection for purposes of inspection by the pathologist. Every now and then, the finding of a polyp or even a small flat independent cancer, undetected by sigmoidoscopic examination, may reward the surgeon for the observance of a caution which by some may be regarded as unnecessary. Such findings suggest the necessity for continuing the excision of segments proximally until no further lesions are encountered. And when one is contemplating anastomotic operations the proximal third of the transverse colon is a good stopping place, even when the anastomosis is to be made to the mid-rectum.

In this connection an interesting report of Mayo and Schlicke[6] of an autopsy study of polyps in the colon is of some importance. In patients coming to autopsy in whom the primary lesion was cancer of the colon or rectum, an additional polyp or polyps were found in 34.1 per cent of all patients. Moreover, an independent unsuspected cancer was found in 4.1 per cent

of instances. In patients dying of disease other than primary cancer of the colon or rectum, the incidence of polyps was 14 per cent. The incidence of polyps reported in the Mayo Schlicke study is considerably higher than is reported generally in barium and proctoscopic studies. In examinations in living patients, such unsuspected polyps are found ordinarily in 2 to 7 per cent of instances. Inasmuch as the polyp is a frequent precursor of colic or rectal cancer, it is certain that additional unsuspected polyps are more likely to be found in patients who come for the treatment of rectal or colic cancer. In any case it behooves the surgeon operating for malignancy of the colon or rectum to beware lest he overlook a precursor of a malignancy or an already existing independent cancer. If the Mayo and Schlicke observations are borne out in similar future studies, the surgeon who undertook to extirpate regularly the entire left colon, the area in which polyps are found most frequently, for lesions involving either the rectum, rectosigmoid or sigmoid colon, could probably justify his rashness by the finding of unsuspected potential malignancies or already existing independent cancers in sufficient numbers to justify the extra labor. Swinton and Haug[11] report upon a similar autopsy study of 1,843 patients. They observed an incidence of benign polyps in 7 per cent of these patients. In 42 per cent of the patients with polyps two or more benign lesions were present.

EARLIER RECOGNITION

Whether we deal with the point of entry or exit or intermediate conduits or reservoirs in between the portals of the alimentary tract, we all know how very important is early diagnosis for the successful management of cancer. Much as we lament the failure of patients to heed the warning of blood in the stool, pain or slight change in the bowel habit with reference to cancer in the lower reaches of the alimentary tract, yet an even more primary concern is that visceral cancer is a silent

disease. The length of time involved in the transformation of normal epithelial cells from a normal mucous membrane into malignant cells is not known; how long it takes before invasive features cause the appearance of symptoms is undoubtedly a variable period, perhaps usually in excess of a year. Inasmuch as cancer of the alimentary canal including the colon and rectum is frequent and because cancer is an insidious and silent disease, we should take the pains to carry this instruction to the public; moreover, we should implement means of enlisting the public's concern and co-operation in thwarting the menace of latent cancer.

Whereas visceral cancer may occur at any age, the majority of cases of cancer of the colon and rectum are observed in patients beyond fifty years; in this series of rectal cancers, the extremes of age were thirty-one and eighty-six years; only 10 per cent of the patients were under forty-five years. In gastric cancer there appears to be a sharp rise of such cancers in the male at approximately fifty years of age. In colic and rectal cancers this rise in incidence appears to occur somewhat earlier. Until more specific means of detecting the presence of cancer are available, our only hope for more consistent early recognition of cancer is the routine examination of populations in which cancer may be developing by precise technics of examination. Such pilot cancer detection centers will, we believe, establish the validity of the thesis that silent cancer can be diagnosed. That many adults will have cancer is a foregone conclusion. It is merely a question of *who* will have it, in *what* organ and *when*.

We are concerned over making a more important impact upon the important problem of rectal and colic cancer than our present accomplishment suggests. It would be eminently fair to say here that the labor and the cost of operating a cancer detection clinic would be fully justified on the score of the digital and endoscopic findings attending examinations of the rectum and

lower sigmoid alone. Future discoveries may probably make the labors of such specialized routine examinations less necessary.

We must be realistic, however, and deal with situations as we find them. The surgery of malignant disease of the rectum and colon can be done with little risk. The only promise for more enduring results is that a larger number of patients come to operation when the lesions are silent and curable by surgery. When that time comes, the conservative operation which salvages sphincteric function will have an even more important rôle than it has now in the management of malignancies in the middle and upper segments of the rectum.

LOW ANASTOMOSIS FOR EXTRARECTAL MALIGNANCIES AND MISCELLANEOUS CONDITIONS

Some of the most difficult surgical problems were constituted by a miscellaneous group of patients necessitating colorectal resection, in whom restoration of intestinal continuity was re-established. Some of these fell in the upper group (14 to 20 cm.) and some in the lower (0 to 13 cm.). There were two patients with malignant tumors of the ovary involving the rectum and uterus or vagina. One of these patients when weighed directly following the excision of the enormous tumor was 15 pounds lighter despite liberal hydration and generous administration of blood during a long operative procedure. This tumor had been explored twice elsewhere and had been said to be inoperable. In addition to the rectal resection, a jejunal and bladder resection also were necessary in this individual. The colorectal anastomosis fell in the higher group. The other patient had undergone a previous right colectomy for cancer and a subsequent subtotal hysterectomy. In this instance the cancer of the ovary involved the rectum, cervix and upper third of the vagina. The colorectal anastomosis was made 5 cm. from the anus; the wound healed by first intention without sinus or

fistula formation. It was looked upon as a palliative procedure but she has remained well for three years since operation with excellent sphincteric function. In one patient with a huge cancer of the cecum a fistulous communication existed with the rectosigmoid necessitating two simultaneous anastomoses. The lower colorectal anastomosis belonged to the upper group and the ileum was anastomosed end-to-end to the transverse colon. Table III lists the cases. An additional patient bears mention. She had tuberculous peritonitis and a colostomy had been performed in 1941 because of a stricture in the lower gut. At a subsequent operation an extensive tuberculous peritonitis of the pelvic colon was encountered. Considerable necrotic material in juxtaposition to the pelvic colon was curretted away, two simultaneous intestinal resections being carried out in the right and left colon. A stricture persisted at 10 cm. from the anus and ultimately a low anastomosis was carried out with subsequent closure of the colostomy. Inasmuch as the colostomy was of the Devine type, the only such colostomy ever made in our hospital, the closure had to be effected by excision of the colostomy segments and end-to-end anastomosis. Just as effective fecal deviation can be achieved by a single loop colostomy.[14]

PRIMARY RESECTION FOR CHRONIC ULCERATIVE COLITIS

There is a small group of patients with chronic ulcerative colitis for whom a primary resection can be done. Amongst the operations listed herein, there were thirteen such patients. The operations performed are listed in Table XII and the distribution of the disease is outlined in Table XIII. That such resections can be carried out in patients with segmental distribution of ulcerative colitis is understandable; a number of the patients with ulcerative colitis had that type of lesion. (Table XIII.) However, there were two patients in the group in whom the lesions of ulcerative colitis were present through-

out the colon and rectum, in which anastomosis of the ileum to the terminal pelvic colon, accompanied by simultaneous excision of the remainder of the colon, was followed by healing of the lesions in the rectum. Obviously, the condition of the

alled by an early disappearance of procto-scopic evidence of the disease from the lower rectal segment.

This experience suggests that in suitable instances of ulcerative colitis, at an earlier stage in the disease, subtotal colectomy;

TABLE XII

CHRONIC ULCERATIVE COLITIS

SURGICAL THERAPY WITH PRESERVATION FOR THE RECTUM OR A PORTION OF IT

January, 1942 to June, 1947

Surgical Procedure	No. Cases	Per Cent Mortality
A. Resection and Anastomosis		
1. Subtotal colectomy and ileocolostomy in terminal pelvic colon..	4	0
2. Subtotal colectomy and ileoproctostomy..........	4	0
3. Partial colectomy and ileocolostomy left colon........	1	0
4. Right hemicolectomy and ileotransverse colostomy.........	1	0
5. Subtotal colectomy and cecocolostomy in terminal pelvic colon*............	1	0
Total..........	11	0
B. Total colectomy and pull-through of ileum............	2	0
Total of All Cases.........	13	0

* This patient was operated upon in 1940.

TABLE XIII

CHRONIC ULCERATIVE COLITIS

Distribution of Diseased Areas

Site of Disease	No. Cases
1. Confined to rectum..................	1
2. Confined to colon..................	2
3. Colon and rectum..................	3
4. Colon and terminal ileum..................	6
5. Rectum, colon and terminal ileum......	1
Total..................	13

with anastomosis of the ileum to the terminal pelvic colon or rectum, may lead to a disappearance of the residual lesions from the rectum.

One patient with a segmental type of lesions involving the colon as far distally as the sigmoid was quite febrile at the time of operation and had been so for weeks; subtotal colectomy was followed by immediate subsidence of fever and general improvement.

Two patients in the group had involvement of the entire colon with a stricture in the rectum at 10 cm. from the anus, with a normal mucosa in the distal rectal segment. Colectomy with anastomosis by suture to the distal rectal segment was carried out in these two patients with a very satisfactory result. Both patients are young women and despite the circumstance that the bowels move three or four times a day, they have excellent continence and bear this burden with no complaint.

In two patients presenting evidences of complete destruction of the large areas of the mucosa of both colon and rectum, a complete colectomy and partial rectectomy together with excision of the rectal mucosa from the distal segment was carried out, pulling the ileum through at the anus. The first of these operations was done on a young school boy five years ago, but the ileostomy has been closed only a few months. Despite quite satisfactory con-

mucosa of the distal rectal segment must be such that there is a possibility of recovery of its function after elimination of the greater portion of the disease. It had been suggested that the making of an anastomosis under such circumstances would probably lead to the upward migration of the disease from the remaining rectal segment into the lower reaches of the ileum. As a matter of fact, the reverse occurred. The disease in the rectal mucosa cleared up. In one instance this clearing process was slow as was indicated by the persistence of a tendency for the mucosa to bleed on proctoscopic examinations and by the continuance of diarrhea (four to six stools a day). In the remaining patient the improvement in general health was par-

tinence, the bowels move every two hours day and night because of the diarrheal character of the stool. Obviously, if this situation continues, the boy would be better off with an ileostomy bag.* In the other patient, the ileostomy has not yet been closed.†

In order to avoid impotence which quite often attends performance of the abdominoperineal operation for excision of the rectum in the male, colectomy and partial proctectomy with excision of the mucosa from the distal rectal segment has been carried out in one patient (not in this series) with simultaneous establishment of an ileostomy. The abdomino-anal pull-through operation for ulcerative colitis described above was proposed for this patient but he rejected it. This operation done more than five years ago was followed by a very satisfactory result with preservation of a normal sex function.

SUMMARY AND CONCLUSIONS

The experience of this clinic with restoration of intestinal continuity after excision of primary cancer of the rectosigmoid and rectum has been reviewed. The following conclusions seem warranted:

1. For all lesions in the rectosigmoid area 14 to 20 cm. from the anus, the conservative operation affords the patient as satisfactory a prospect of cure as does the abdominoperineal operation. In any case, no local recurrences have been observed after resections for cancers at this level.

2. For lesions in the lower rectal segment, at 8 cm. or less from the anus, the conservative operation is not a good operation for primary rectal cancer, primarily because it does not deal effectively with the invisible lateral spread of cancer as does

the more radical abdominoperineal resection. In low-lying lesions a fairly high incidence of local recurrence stongly suggests that salvage of the rectal sphincters is accomplished at the risk of failing to cure the cancer.

3. For suitable lesions in the mid-rectal segment, above 8 cm. from the anus, the conservative operation would appear to be as satisfactory as the abdominoperineal; however, for fixed lesions, the abdominoperineal operation, undoubtedly the most radical operative procedure available, offers some advantage, the extent of which cannot yet be completely assessed.

4. The experience with the low anastomosis also is reviewed in a series of extrinsic rectal tumors as well as in a miscellany of other indications in which the lesion together with a portion of the rectum was excised with re-establishment of intestinal continuity.

5. The experience with primary restoration of intestinal continuity after excision of subtotal lengths of the colon and rectum for a small, select group of patients having chronic ulcerative colitis is recounted.

6. Finally, it may be said that satisfactory rectal continence attending these operations, particularly the suture methods of restoring continuity, is quite uniform with consistent preservation of a normal sex function. The importance of preservation of the internal sphincter for the maintenance of fecal continence cannot be overemphasized. A satisfactory anastomosis made by the suture method 3 to 4 cm. above the pectinate line assures complete continence.

* Since this was written, this patient has undergone vagotomy with considerable improvement. Vagotomy was done for two reasons: (1) to slow the gastric emptying time, (2) to attempt to abolish the gastro-ileac reflex.

† Since this paper was written, Drs. Ravitch and Gabiston of Baltimore have described carrying out such a procedure on the dog.[8] Their article is also well illustrated. The procedure was described briefly by one of us four years ago.[12]

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DISCUSSION

THOMAS E. JONES (Cleveland, Ohio): I would like to say that unfortunately too many people in this country today think that the anastomotic operation is something new. Much has been written about it without reference to things in the past and the younger men at any rate think that this one-stage operation is a new operation. Well, I looked over my figures for the past twenty-seven years since 1920 and I am surprised at the number of one-stage end-to-end anastomoses that I made then and without complementary colostomy. Unfortunately, many of the people who are writing about this subject put complementary colostomy in fine print down below. I say that because people have come to my clinic and asked me if I do the primary resection. I said, "What is your definition of a primary resection?"

"Well, just cutting it out and sewing it together and dropping it back and that is the end of it."

Well, that is fine. I did it many, many years ago, but I changed to something that I believe is safer and now I do the great majority of operations by the exteriorization method. The reason that I did the end-to-end anastomosis in those days was this: I had seen many cases of local recurrence in the abdominal wall following a Mikulicz procedure. Therefore, I would not do one. I did the end-to-end anastomosis with or without tubes with the colostomy above.

Then I came across the Rankin modification of the Mikulicz which appealed to me. However, I believed that such a procedure with the quoted mortality at that time was too high and, therefore, with the mortality of 12 per cent in those days for end-to-end anastomoses, I tried the Rankin resection modification of the Mikulicz procedure because I thought that I could lower it. I have been able to lower it. I will continue to do it because in not a single case I did have I had a recurrence in the abdominal wall. What does that mean? It means that all the gland-bearing area is removed. The trouble with the old Mikulicz operation and even the Rankin resection operation is that the mesentery obviously is stuck up against the peritoneum of the abdominal wall when you close the colostomy. If you are there when you do the operation. It is going to have local recurrence in the mesentery, you are going to have local recurrence in the abdominal wall. I have not seen a single case of my own series which means to me that I can do a more radical operation with this procedure than I can with the end-to-end anastomosis in a good many segments because I do not have to worry about the blood supply. In the past, patients with peritonitis did not die from contamination following an open operation; they died from necrosis at the suture line. If I do not have to worry about that, I believe that I am going to have a lower mortality.

It seems to me in looking over all the evidence that the only controversy is about what level you are willing to do an anastomosis. Knowing what to do about malignancy, it does not make any difference.

Dr. Wangensteen started out a few years ago believing surgery could be done with lesions within 4 cm. of the anus. I think the last thing that he said was 8 or 10 cm. I believe that before very long he will say 12, 15 or 16 cm. Those things were done in the old days at that level. For that reason it is nothing new.

What is new about it is anastomosing them below the reflection of the peritoneum and that is distinctly new. I do not have confidence in the operation. Of course, it can be done technically, but I believe that one must do an awful lot of them before you can do them technically. If you take the rank and file of all ages and all weights, it is an extremely difficult operation. It is a far more difficult operation than the abdominoperineal resection.

I came to do the radical operation and the Miles operation because I was brought up in a small way also. Readers are acquainted with the operations that were in vogue in 1915 to 1925. They were small operations. The Kraske operation, the pull-through operation; everything was done to try to save the sphincter. I would rather have a patient alive and well than have him have a local recurrence. As Miles often said, "The patient has been sitting on this cancer too long before he comes to me, and I am not going to contribute to him sitting on it any longer."

I am sure that the smaller operations are bound to lead to more local recurrences. As a matter of fact, there is a paradoxical figure here. David and Gilchrist, three months ago, showed figures in which their local recurrence was 20 per cent. Now, it is something new in malignancy and our conception of malignancy if we are going to have a lower local recurrence from a smaller operation. There is something wrong with the figures. Of course, I think, as Dr. Wangensteen has said, the time is too soon and the number of cases too small to draw final conclusions. It is an admirable attempt, however, to improve the situation and for that we give him credit. However, in the meantime one must use his own judgment as to what operation fits him best and the number of patients that he thinks he will be able to benefit.

I would say that if the percentage of recurrence, which I think is going to be higher with these smaller operations, is going to be higher than with the abdominoperineal operation, that alone is sufficient reason to do the abdominoperineal resection because there is little use in transplanting one morbidity with another? A man with cancer in situ in the rectum is ten times better off than one with a perineum which finally becomes necrotic from broken down malignancy.

In his paper Dr. Wangensteen reported that in certain lesions of the descending colon he may go up and remove the entire descending colon, the splenic flexure and so forth and anastomose the transverse colon to the rectum. He wrote that a man who does the abdominoperineal operation and the Mikulicz operation cannot stand on the same stage. I certainly cannot see how a man can stand on the same stage and be satisfied with getting 2 cm. below a low rectal growth and yet remove a foot or two of colon which is so absolutely and absurdly unnecessary because you never get glands up in the splenic flexure from a growth in the sigmoid or the descending colon. The vulnerable area, of course, is the gland, and there is just so much you can do; and if you tamper too much to get those glands out, you are going to have a very high mortality.

The mortality, as we know, has been brought down. This operation started in 1925 with Miles, and the mortality was 25 to 30 per cent. In this country it was lowered to 20, 15, 12, 10, 7, and so forth. That has been done by a combination of factors that I do not have to enumerate here. You know what they are: better preoperative treatment, better anesthesia, better care of the patient afterward, at least until the war emergency, all of those things. You cannot point to one factor which made the mortality less. Of course, I am small minded enough to believe that the only thing that has made a difference in my series is the use of steel wire sutures. Of course, everybody laughs at it, but that is just my fetish. I believe in my old experience and my new experience that it has immensely cut down mortality.

Today we have a series of 535 consecutive cases from 1942 to 1946 of abdominoperineal resection in one stage with twenty deaths, or a mortality of 3.7 per cent. Therefore, mortality is a thing of the past, I believe, with present-day good hands and good hospital facilities.

Ten of these deaths were from embolism, many of them on the day they were going home. You all know that story. But the one thing I would like to ask you is in connection with this embolism mortality and what is being advocated in Boston. Would you be willing to ligate 1,070 femoral veins in order to try to offset ten deaths from embolism? I do not know that I would at this time, because I am not absolutely sold on the idea of femoral ligation, to do it prophylactically as advertised in all malignant cases. I do not believe that I would

choose to do it in contemplating another series like this in the next five years.

Table 1 shows a group of cases from 1940 to 1944; 117 resections were done by the Mikulicz technic, with six deaths. When you do 117 of these cases in the sigmoid, you know what you

TABLE I

CARCINOMA SIGMOID OBSTRUCTIVE RESECTION

Year	Res.	Mort.	%
1940	16	0	
1941	21	3	
1942	23	1	
1943	22	0	
1944	35	2	
Total	117	6	5.1%

TABLE II

ABDOMINO-PERINEAL RESECTION

Year	Cases	Mort.	
1942	73	0	
1943	91	5	
1944	124	6	
1945	112	6	
1946	135	3	
Total	535	20	3.7%

are dealing with. Pieces of the bladder have been removed, the uterus may be removed, the adnexia have been removed, oftentimes a very large portion of the peritoneum is removed. Many of those cases are perforated at the time you get them, so that it seems to me the sigmoid is really by and large a bigger problem than the rectum for these complications. A mortality of 5 per cent in 117 cases is perfectly satisfactory to me because of the type of cases that they are done on and we have no local recurrences.

Table II demonstrates our group of 535 cases; there were 137 consecutive cases without a death. This was entirely too long, of course, and your luck cannot go that way all the time, as Dr. Wangensteen has pointed out. At any rate the series was a long one and we were interested in attacking it from all sides to see if we could find any one factor which was responsible.

I think we know a little bit more about cancer than we used to certainly; we do not know half

enough. The glandular dissections and the information we have had from that is very important. However, I think the venous involvement is ten times more important than the glandular involvement.

In a group of 103 consecutive cases studied about ten years ago after the method of Duke and David and Coller, and so forth, we found 65 per cent of the glands involved. Then after reading some articles on pathology, particularly by Warren, we investigated this same group of cases again and without staining for elastic tissue, Dr. Graham was able to demonstrate venous involvement in 72 per cent of the cases, and he predicted that if he would stain all of these for elastic tissue to see where the malignant cells were, that he would probably do it in a higher percentage of cases. What does that mean? When you do a low anastomosis, you must tear through the mesentery fat. You may get 2 cm. below a good mucosa, but I believe the trouble is in the surrounding tissues, in the surrounding fat. Dr. Graham, I must say, laid more emphasis on invasion of the mesenteric fat as a prognostic thing than glandular involvement. I believe that it is true. The venous involvement is the important one. I believe unless you get very far around the growth, you are going to get local recurrence from these cells in the veins.

JOHN M. WAUGH (Rochester, Minn.): In attempting to cure malignancy by means of operation three important factors should be given consideration by the surgeon: First, does the operation he proposes to use carry the best chance of cure for the patient of any of the procedures available; second, is the risk of operation reasonable or does it outweigh the chances of cure; and third, is the patient left in a normal condition following surgery and able to carry on his work without impedient or embarrassment. Because in the past so many patients have of necessity had permanent and temporary colostomies following operations for cancer of the colon and rectum, it is fitting and timely that we periodically review our operative procedures to see if more patients cannot be spared colonic stomas without sacrificing a high curability rate or a low operative mortality.

In the past ten years we have seen the staged procedures for resection of the abdominal colon gradually give way to the single stage operation without temporary colostomy except in the

faee of obstruction. Right hemieoleetomy has been done in one stage at the Mayo Clinic for several years with immediate primary anastomosis with a mortality of 2 to 3 per cent. In 1946 eighty such resections were earried out without a hospital fatality. In the transverse colon and descending colon to the lower sigmoid resection with primary anastomosis has been done, as Dr. Wangensteen has shown, with a mortality of 5 per cent and less. In the past five years on my own service 197 resections with primary anastomosis have been done in the left colon with a hospital mortality of 5.5 per cent. This figure includes resections for carcinoma of the rectosigmoid, upper rectum and lower sigmoid. If one considers only the group of lesions that could have been ex-

teriorized and excludes the lower group, there were 117 patients with a 4 per cent hospital mortality. I do not believe that this mortality eould have been lowered by use of the exteriorization procedure. The length of hospital stay for the single stage procedure has averaged one-third that of the multiple stage. This is no small economic factor, especially if one elects to carry out palliative resections for obviously incurable disease.

Most of the controversy in recent years concerning rectal surgery has been (1) Is the sphincter conserving operation radical enough for cure whether it be done by anterior resection as advocated by Dixon and Wangensteen or by the pull-through procedure popularized by Babcock and Bacon? (2) Is the operative

TABLE I
INCIDENCE OF RETROGRADE NODAL METASTASIS IN CARCINOMA OF RECTUM AND RECTOSIGMOID: REVIEW OF LITERATURE

	Total Cases	Positive Nodes (Cases)	Nodes Involved below Lesion (Cases)	Distance below Lesion	Site of Lesion
Westhues..................................	74	?	1	1	?
McVay, 1922..............................	100	47	1	0–1 cm.	Ampulla
Wood and Wilkie, 1933......................	100	51	0		
Gabriel, Dukes and Bussey, 1935.............	100	62	2	0–1 cm.	Upper rectum (8 and 9 cm.)
Gilchrist and David, 1938....................	25	16	2	−2 cm. −4 cm.	Ampulla
Coller, Kay and MacIntyre, 1940.............	33	22	1	1.5 cm.	Rectosigmoid
Grinnell, 1942.............................	75	41	1	1 cm.	Ampulla (6 cm.)
Total..................................	507	239(+)	8		
Glover, 1944..............................	100	100	36		Upper rectum
			27	0–1 cm.	
			6	1–2 cm.	Rectosigmoid
			1	2–3 cm.	
			1	3–4 cm.	Lower sigmoid
			1	6–7 cm.	

mortality low enough and the functional result sufficient to justify these sphincter conserving operations?

First, as far as cure is concerned with these operations, I am satisfied personally that they are just as curative as the Miles procedure for

TABLE II
ANTERIOR RESECTION (NON-PALLIATIVE OPERATION):
FIVE-YEAR SURVIVAL RATES FOR DIFFERENT LEVELS
(Dr. Dixon's Data)

Distance from Dentate Line, Cm.	All Cases		No nodal Involvement		Nodal Involvement	
	Number	5 Yr. Survival Rate, %	Cases	5 Yr. Survival Rate, %	Cases	5 Yr. Survival Rate, %
6–10	74	63.7	32	72.4	42	57.1
11–15	97	70.2	58	78.8	39	57.7
16–20	101	66.9	60	71.5	41	60.4
All Levels	272	67.7	150	74.0	122	58.5

lesions where the lower edge of the cancer is 5 cm. or more above the pectinate line. The work of Glover on retrograde spread (Table I) predicted such would be the case and this has been substantiated by Dr. Dixon's five-year cure rate following anterior resection. (Table II.) Second, the operative mortality of anterior resection is approaching that of the Miles operation but because of the suture line undoubtedly will always be a trifle higher even in the hands of those with considerable experience. The pull-through procedure, however, can be done with just as low a mortality as the Miles and in my own experience of approximately one hundred there has been one hospital death. Anal control after even low anterior resection has been excellent. Following the pull-through operation most patients do not have perfectly normal control and few are able to control flatus. The majority, however, rarely soil themselves unless diarrhea appears and all patients prefer the procedure, even those with the most unsatisfactory results, to an abdominal colostomy.

OWEN H. WANGENSTEEN (closing): All of us in a sense are surgical Fausts. I am not thinking of those who sell themselves for thirty pieces of silver or a mess of pottage. I am talking about compromises which surgeons are willing

to make. Dr. Jones would have us believe that he affects less interest in preservation of fecal continence and sexual function than he does in the contour of the female breast. That is an interesting attitude of mind. Dr. Jones apparently finds no difficulty in defending the necessity of performing the abdominoperineal operation in all patients with cancer of the iliac colon and rectosigmoid in which the Mikulicz procedure cannot be done, an admission suggesting perhaps that he is a better master of the abdominoperineal operation than he is of primary resection. Certainly the time has come for experienced surgeons to exteriorize the exteriorization operation in dealing with cancer of the colon. In practiced hands, the two operations can be done at the same risk with an important salvage in hospital stay and expense to the patient for whom primary resection is done.

I grant freely that I have overextended myself in the anxiety to please a patient who wishes her sphincteric function saved. Like the young lady in the musical comedy "Oklahoma", I have found it difficult now and then to say no—but I am learning.

With reference to the recurrences which we have observed after the anastomotic operation, it perhaps is not out of place to emphasize that these have not been in the bowel wall primarily; on the contrary, the recurrences first made their appearance outside the bowel involving it secondarily. In other words, what we interpreted as a Dukes' group A case, on occasion belonged in reality to Dukes' group C, in that, invisible lateral spread already had occurred at the time of the initial operation but had not been identified as such. Our own experience suggests quite definitely that for the upper group (14 to 20 cm. from the anus) the anastomotic operation holds out just as much promise of cure as does the abdominoperineal operation. For patients with lesions less than 8 cm. from the anus we have given up the anastomotic procedure because of the greater hazard of local recurrence than follows the abdominoperineal operation. The controversial group, it seems to me, is the intermediate group between 9 and 13 cm. from the anus. There is no debate about the superiority of the abdominoperineal operation for this group; that is granted. The question is: How much better is it from the standpoint of curability of cancer than the anastotmotic pro-

cedure? This is the group in which some case selection may have to be done. Among thirty-two patients with lesions at this level for whom an anastomotic procedure was carried out, local recurrence was observed by us twice (6.3 per cent). With proper case selection I believe that the anastomotic procedure with satisfactory removal of the lymphatic drainage area can be done in a large number of patients with lesions at this level.

Dr. Waugh's figures are excellent. In fact, they are so good that one cannot help but conclude that some selection must have occurred in his groups.

With reference to the kind of anastomotic operation to be carried out our experience suggests definitely that the direct suture carried out as a one-stage procedure from the abdomen is the best operation. For anastomoses made at 8 cm. or more from the anus drainage is rarely necessary and a supplemental external decompressive vent is not necessary. With elimination of the anastomotic procedure for lesions lying less than 8 cm. from the anus the single row direct suture method becomes, save for a few exceptions, the standard procedure.

A young woman will put up with less complaint on this score, getting rid of an abdominal ileostomy, than will a young man. I know young marriagable girls who had an ileostomy for some time and they are closed. Even though they have four or five stools a day, they make no complaint about it. I suppose maybe the male is a more complaining creature. He will suggest two or three stools a day is a burden for him to bear.

Will you read the questions and I will try to answer them one by one, if I can.

Dr. Ault: Doctor, I understand this is in a research process. I am wondering how many anastomoses between ileum and involved rectum or rectosigmoid in cases of ulcerative colitis have held up for how long.

Dr. Wagensteen: Obviously, an attempt to conserve the rectum or part of it in the type of case demonstrated by Dr. Ault on the screen would be foolhardy. If one is going to anastomose the ileum to a residual rectal segment, that segment should be free of ulcerative colitis or have only punctate hemorrhages in the mucosa without evidence of destruction of the mucosa.

Dr. Ault: Thank you, Dr. Wangensteen. We want to make this clear to everybody. How many have had to be converted back to ileostomy?

Dr. Wangensteen: That is easy to answer —none.

Dr. Ault: What complications, stricture, abscesses and what morbidity have attended this procedure of anastomosing the ileum to involved rectum or rectosigmoid?

Dr. Wangansteen: There has been no mortality either accompanying operation or since operation. Whereas some of the patients with mild evidences of disease in the distal rectal segment have continued to have some diarrhea, it has been gratifying to observe that extirpation of 85 to 90 per cent of the extent of the disease has permitted the lesions in the residual rectal segment to improve. Moreover, migration of the disease into the ileum after such anastomoses has not been observed. Only one patient has been operated upon in a febrile state. To that patient we gave streptomycin, sulfasuxidine and penicillin prior to operation. A one-stage operation was done anastomosing the ileum to uninvolved rectum, excising the involved colon between. The patient convalesced uneventfully and left the hospital about a week after operation.

Some of the matters discussed today obviously are controversial in nature; however, we cannot resolve our differences by debate but rather by analyzing our experiences critically and recording them truthfully. That is what Dr. Toon and I have tried to do in this paper. Time is the final arbiter of all things. Until she has given her answer, let us be tolerant of our honest differences of opinion.

POSTERIOR LEVATOR SPACE ABSCESS*

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DEEP perirectal abscesses are of much concern to the surgeon. It is commonly believed that they occur within three deep perirectal spaces (right pelvirectal, left pelvirectal and retrorectal) and also within the wall of the rectum (intramural). On many occasions these abscesses remain unrecognized and progress for an indefinite period, thus permitting relatively simple abscesses to become complicated by spreading from one deep space to another, or by rupturing high into the rectum, causing that serious complication, the true "anal-rectal" fistula. The pathways by which infection spreads from the anorectum to these deep spaces never have been accurately understood or established on a strictly anatomic basis. My dissections of the pelvic diaphragm and its related structures reveal the presence of a fourth deep space, lying posterior and lateral to the anorectum and situated between the superior and inferior layer of the levator muscle. This space is apparently undescribed; to refer to it, I shall use the term, "posterior levator space."[1,2] In my clinical experience this space frequently is involved by a deep perirectal abscess.

RESEARCH MATERIAL

Pelves of eight human cadavers of the Negro and white races, male and female, were dissected in detail. Pelves of forty cadavers of the Negro and white races, male and female, were surveyed in the laboratory while dissected by others. This description is based upon a dissection made from both the perineal and pelvic approach and a comparison of the dissection with sagittal, parasagittal and coronal sections.

TERMINOLOGY

In clinical literature on this problem various terms have been applied to indicate certain useful anorectal landmarks. These have not always been identified on a strictly anatomic basis by their originators; hence, others using these terms have frequently introduced confusion into the field. In some instances the inventors of the terms may have been confused themselves. For clarification, I shall define and limit the terms to those in use at the Philadelphia proctologic clinics, indicating the specific anatomic entities intended.

True Anal-Rectal Fistula. This is a very rare type of fistula; one in which the primary opening occurs in one of the crypts along the anorectal line and the secondary opening, high in the rectum.

Anorectal. This term is an adjective used to refer to the general region of the anus and the rectum. When referring to an anorectal fistula, it means a fistula in which the primary (internal) opening is either in the anus, at the anorectal line or in the rectum. It does not mean that the primary opening is in one and the secondary opening is in the other.

Anorectum. This noun refers to the general region of the anus and the rectum.

Combined Longitudinal Muscle Layer of the Rectum. This phrase describes that thickened portion of the longitudinal muscle layer of the rectum which extends
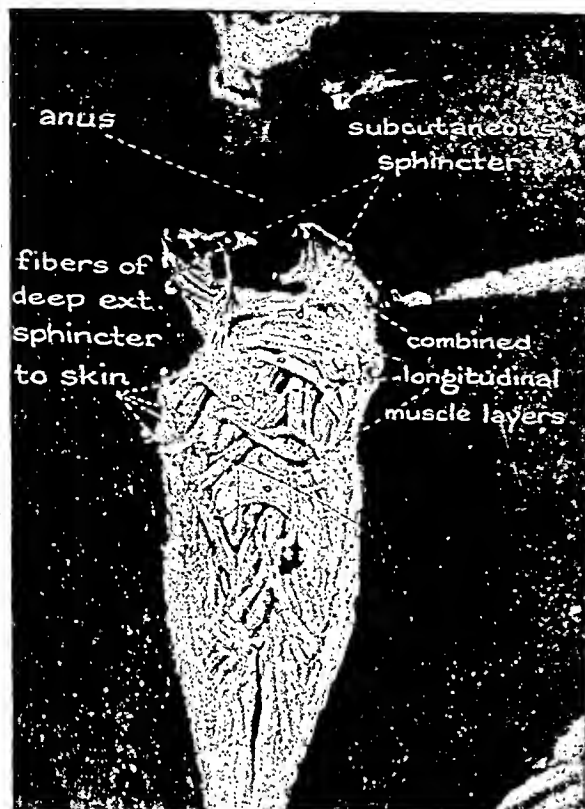
FIG. 1.* Retouched photograph, showing the insertions of the subcutaneous sphincter, the deep sphincter and fibers from the combined longitudinal muscle layer of the rectum, into the skin along the posterior midline. An incision has been made through the skin in the midline (anoeoecygeal skin sulcus) and extended into the anal canal. The skin edges have been pulled apart and held by pins. (Dissection by the author.)

inferiorly from the level of the levator fascia to the perianal skin below. It is composed of the longitudinal muscle layer of the rectum, fibers from the levator fascia and muscle fibers from both the superior and inferior layers of all three portions of the levator muscle (puborectalis, pubococcygeus and iliococcygeus).

Levator Fascia. This is the fascia covering the superior surface of the levator ani muscle.

Iliorectococcygeus Muscle. This is a new anatomic term proposed for the rectococcygeus muscle because this muscle is

* Due to the fact that the relationships of a single structure may be mentioned in several different locations, Arabic numerals have been used in the text to indicate each specific relationship in its various locations. Thus (III-9) as it appears in the text, refers to the "retrorectal space" (9) on Figure III.

formed mainly by fibers from the iliococcygeus (striped), fibers from the longitudinal muscle layer of the rectum (smooth) and a few fibers from the levator fascia.

Circumanal Space. This is another new term proposed for a space which encircles the anus. It lies between the inferior edge of the internal sphincter muscle and the anal intermuscular septum. Its inferior boundary is formed by those fibers from the combined longitudinal muscle layer which form the anal intermuscular septum and insert into the skin of the anal canal. Some of these fibers also pass upward, medial to the internal sphincter, to insert into the anal skin at a higher level, thereby forming a "U-shaped" sling around the inferior edge of the internal sphincter.

Parietal Layer of the Pelvic Fascia. This refers to that portion of the pelvic fascia surrounding the muscles, blood vessels, nerves and lymphatics of the pelvis.

Coccygeal Muscular Raphe. This is a proposed new term to indicate more accurately the musculo-fibro-tendinous insertions of the inferior layer of the levator (puborectalis, pubococcygeus and iliococcygeus) into the tip and sides of the coccyx; formerly referred to as the "anococcygeal raphe." The term "anococcygeal raphe" is a misnomer since this web of muscle fibers does not attach to or come in contact with the anus in any place.

Anal Fascia. This term refers to the fascia covering the inferior surface of the levator ani muscle and the external sphincter muscles.

ANATOMY

As the individual muscle bundles of the levator pass medially (Figs. II-1 and IV-4), they divide near the rectum into a superior (Figs. II-2 and IV-2) and an inferior (Figs. II-3 and IV-3) layer. These two layers diverge from each other slightly in a wedge-shaped manner (like a letter "V" lying on its side, with the open end toward the midline), thereby forming an anatomic space which is lateral and posterior to the
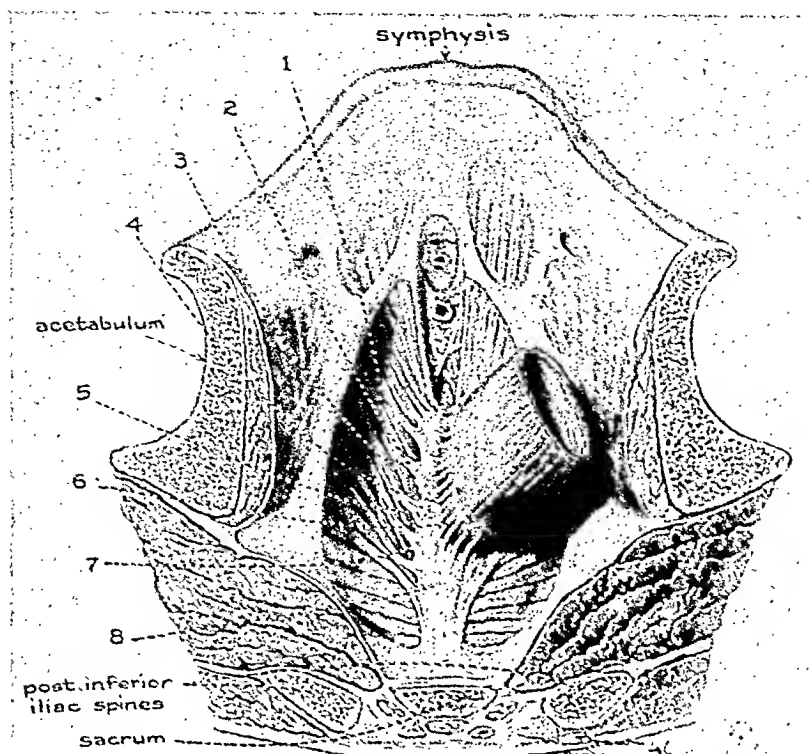
FIG. 11. Drawing showing superior view into dissected male pelvis, the rectum being displaced anteriorly and to the right. (Dissection by the author.) 1, Levator muscle lateral to the point of division into a superior and an inferior layer. 2, Superior layer of the levator. 3, Inferior layer of the levator. 4, The row of fossules with connecting tracts which extend inferiorly to the circumanal space (level of anal-intermuscular septum). 5, Superior layer of levator forming the superior boundary of the posterior levator space to the side of the rectum. 6, Longitudinal muscle layer of the rectum giving off fibers posteriorly, which unite with fibers from the iliococcygeus and fibers from the levator fascia, to form the iliorectococcygeus muscle. 7, The lateral margin of the iliorectococcygeus muscle, the usual point of "break through" for abscesses from the posterior levator to the retrorectal space. 8, Iliorectococcygeus muscle forming the superior boundary of the posterior levator space, posterior to the rectum.

A

FIG. 11. A, Actual photograph showing superior view into dissected male pelvis; as shown in Figure 11. (Dissection by the author.) Only structures on the left side of the pelvis have been dissected.

FIG. II. B, actual close-up photograph showing the manner in which the individual muscle bundles of the levator divide into a superior and an inferior layer. The left edge of the iliorectococcygeus muscle is lifted up by pins. (Dissection by the author.) 1, Individual muscle bundle of the levator muscle (before division). 2, Superior layer of the levator (individual muscle bundle). 3, Inferior layer of the levator (individual muscle bundle). c, showing the row of fossules, lateral and posterior to the rectum. (Dissection by the author.) 4, Row of fossules with connecting tracts which extend inferiorly to the circumanal space. 5, Superior layer of levator forming the superior boundary of the posterior levator space to the side of the rectum.

rectum. This space is against the rectal wall; its internal boundary is the combined longitudinal muscle layer of the rectum (Fig. III-1 and 5) in the male and the layers of the rectum and vagina in the female.

The superior layer of the levator unites with fibers from the longitudinal muscle layer of the rectum (Fig. II-6) and the fibers of the levator fascia to form the iliorectococcygeus muscle. (Figs. II-8, III-8 and IV-1.) The "posterior levator space," (Fig. III-3) lies beneath the iliorectococcygeus muscle, surrounding the rectum posteriorly and laterally, in the form of a horseshoe.

As the superior (Figs. III-6 and IV-5) and inferior (Fig. IV-6) layers of the levator come in contact with the rectal wall, each

D

FIG. II. D, actual close-up photograph showing the manner in which the individual longitudinal muscle fibers of the rectum pass into the iliorectococcygeus muscle. (Dissection by the author.) 6, Fibers from the longitudinal muscle layer of the rectum passing posteriorly into the iliorectococcygeus muscle. 7, The usual point of "break through" for abscesses from the posterior levator to the retrorectal space. 8, Iliorectococcygeus muscle.

layer gives off thin bundles of muscle fibers to the combined longitudinal muscle layer of the rectum. In doing so, they form a series or row of fossules (Fig. II-4) with connecting tracts which extend inferiorly to the circumanal space (Fig. IV-7) [the level of the anal intermuscular septum (Figs. III-15 and IV-8)]. Infection spreads from the anal ducts to these tracts, and thence upward to the various deep perirectal spaces. These tracts lie within the combined longitudinal muscle layer of the rectum. Behind the rectum the tracts to the "posterior levator space" lie posterior to the corresponding ones to the retrorectal space.

Heretofore, we have recognized and defined three deep perirectal spaces, all lying above the parietal layer of the pelvic fascia: (1) the retrorectal (Fig. III-9) space, (2) and (3) the right and left pelvirectal spaces. This paper deals with the discussion of a fourth deep space, the "posterior levator space." (Fig. III-3.)

The superior boundary of this space is formed posterior to the rectum by the iliorectococcygeus muscle (Figs. II-8, III-8

and IV-1) and to the side by the superior layer of the levator. (Figs. III-4 and II-5.) The inferior boundary of this space is formed posterior to the rectum by the coccygeal muscular raphe (Figs. III-7 and IV-11) and to the side by the inferior layer of the puborectalis muscle. (Figs. III-2 and IV-10.)

The deepest part of the "posterior levator space," that is from the top to the bottom, lies posterior and posterolateral to the rectum. As the space approaches the pubis it gradually decreases in depth and width. This space is filled in by a loose layer of fatty, areolar tissue which is readily separated with the blunt probe. Hence, an abscess of the "posterior levator space," as shown by the arrow (Fig. III-3), may spread around the bowel from back to front, and from one side of the pelvis to the other by contiguity.

Due to the fact that this fourth deep space is entirely bounded by subdivisions of the levator muscle and because the most extensive part of this space lies posterior to the rectum, I have given it the descriptive anatomic name, "posterior levator space." (Fig. III-3.)
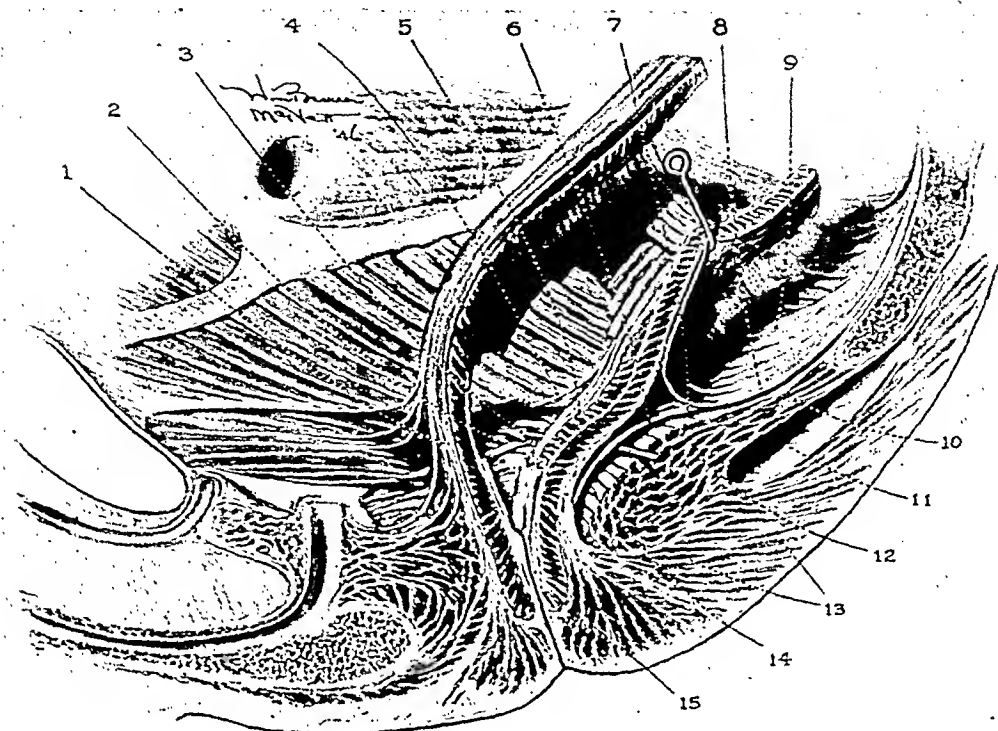
FIG. 111. Drawing of mid-sagittal section somewhat schematic, showing the relationships of the anorectum to the pelvic diaphragm and the anorectal musculature. A window has been cut through the lateral wall of the rectum, layer by layer, to show the relationships lateral to the rectum. (Dissection by the author.) 1, Combined longitudinal muscle layer (anterior to the rectum). This layer is composed of the longitudinal muscle layer of the rectum, fibers from the levator fascia and fibers from both the superior and inferior layers of the levator. 2, Inferior layer of the puborectalis muscle. 3, Arrow lying in the posterior levator space. This space surrounds the rectum like a horseshoe, with the open end of the horseshoe toward the pubis. 4, Superior layer of the levator muscle. 5, Combined longitudinal muscle layer of the rectum (posterior to the rectum). 6, Fiber from the superior layer of the levator, to the combined longitudinal muscle layer of the rectum. 7, Inferior boundary of the posterior levator space formed behind the rectum by the superior surface of the coccygeal muscular raphe. 8, Iliorectococcygeus muscle formed by fibers from the iliococcygeus (striped), fibers from the longitudinal muscle layer of the rectum (smooth) and a few fibers from the levator fascia. This muscle forms the superior boundary of the posterior levator space behind the rectum. 9, Retrorectal space. 10, Coccygeal muscular raphe attaching to the tip and sides of the coccyx. 11, Posterior subsphincteric space (connecting the two ischioanal fossas). 12, Puborectalis muscle (the sling of the puborectalis behind the rectum). 13, Fibers of the deep external anal sphincter muscle inserting into the skin along the anococcygeal skin sulcus. 14, Deep external anal sphincter muscle (posterior to the rectum). 15, Anal intermuscular septum.

CLINICAL MANIFESTATIONS

In all patients observed to date infection began in one of the posterior crypts and extended along the tracts mentioned above to the "posterior levator space."

Infection may be confined to the "posterior levator space" alone but more often there are concurrent abscesses of the retrorectal space or one of the pelvirectal spaces. In some subjects all of the perianorectal spaces, both superficial and deep, were involved. Multiple tracts, emanating from a single crypt, may extend to form simultaneous abscesses in the "posterior levator" and retrorectal spaces.

The extension from the "posterior levator space" to the retrorectal space usually takes place at the lateral margin of the iliorectococcygeus muscle (Fig. 11-7) although this occasionally occurs posteriorly in the midline. Lateral extension between the retrorectal and the pelvirectal space is common, whereas a lateral upward extension from the posterior levator to the overlying pelvirectal space is less common.
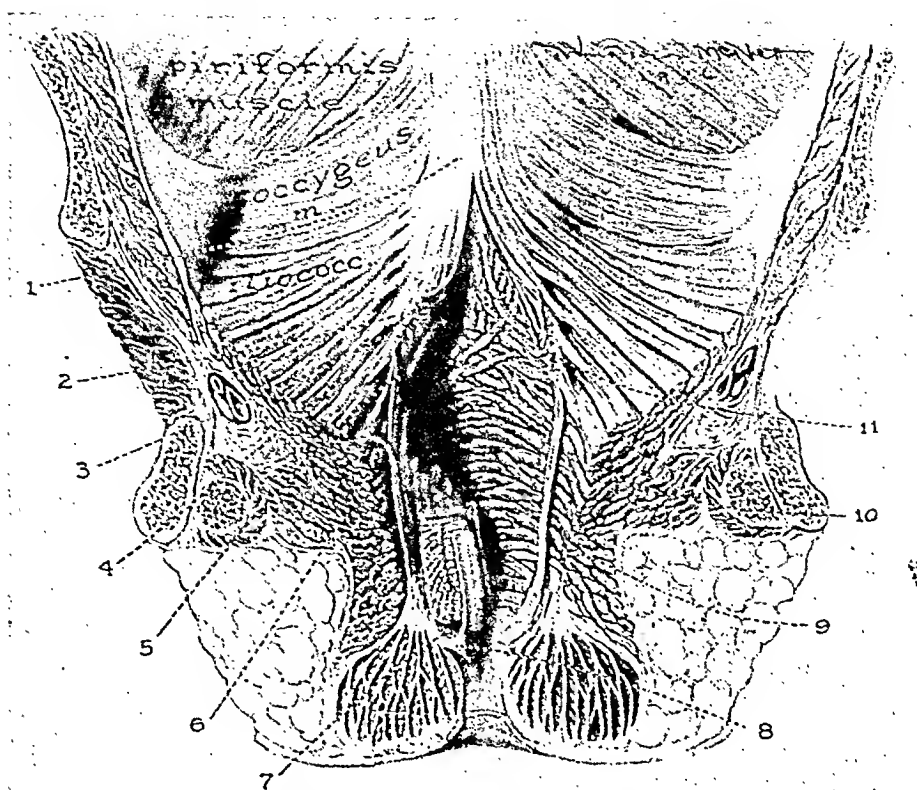
FIG. IV. Drawing of coronal section of dissected male pelvis, somewhat schematic; showing the extent of the posterior levator space posterior to the rectum, and the manner in which the inferior layer of the levator winds itself around the rectum to form the deep external sphincter muscle of the anus. The iliorectococcygeus muscle has been split in the posterior midline and the flaps turned upward. The wall of the posterior half of the rectum has been removed, with the exception of a few muscle bundles of the combined longitudinal muscle layer on each side, and a block of the lower end of the internal sphincter muscle on the right. The skin of the anal canal has been removed. (Dissection by the author.) 1, Iliorectococcygeus muscle. (Superior boundary of the posterior levator space, posterior to the rectum.) 2, Superior layer of the levator. 3, Inferior layer of the levator. 4, Levator muscle (lateral to the point of division into a superior and an inferior layer). 5, Muscle bundle from the superior layer of the levator to the combined longitudinal muscle layer of the rectum. 6, Muscle bundle from the inferior layer of the levator to the combined longitudinal muscle layer of the rectum. 7, Circumanal space. 8, Anal intermuscular septum. 9, Deep external anal sphincter muscle. 10, Puborectalis muscle; inferior layer as it winds and entwines itself around the bowel to form the deep external anal sphincter muscle. 11, Coccygeal muscular raphe (inferior boundary of the posterior levator space behind the rectum).

SYMPTOMS AND DIAGNOSIS

The symptoms of "posterior levator space" abscess are the same as those of any deep perirectal abscess. However, there are several points which help one to make a diagnosis of "posterior levator space" abscess. External evidence of the suspected infection is usually absent, but digital pressure between the tip of the coccyx and the anus produces extreme deep pain. The circumscribed bulge of the abscess into the rectum is readily palpated in the posterior midline. Bidigital examination often reveals the abscess to be under marked tension and protruding into the rectum like a large walnut. In some patients fluctuation is present. Occasionally, the abscess tends to rupture into the rectum laterally, with the bulge occurring in this position. *Due to these facts, abscesses of the "posterior levator space" have been probably mistaken for intramural or retrorectal abscesses in the past.*

SURGERY

In considering surgery several points should be emphasized:

1. Complicated "posterior levator space" abscesses with their inevitable fistulas are often best operated upon in stages. The first stage should include incision and drainage, with location of the primary crypt and tract through which the infection originated. If it is not advisable to remove the primary tract at this time, a seton is placed through the tract, to be removed later.

2. These deep abscesses are opened under direct vision, layer by layer.

3. "Posterior levator space" abscesses are best opened and explored through a posterior midline incision; this separates rather than severs the fibers of the external sphincter and the coccygeal muscular raphe.

4. For counter drainage of the lateral extension of the "posterior levator space," a curvilinear incision is made lateral to the external sphincter muscle. This incision is carried anteriorly to the full extent of the abscess cavity and posteriorly to the lateral margin of the posterior insertion of the external sphincter (which should not be cut). After passing through the ischioanal fossa, the inferior layer of the levator muscle, which is covered by the anal fascia, is identified and incised, thus opening the lateral extension of the "posterior levator space." The skin edges are trimmed to prevent inversion. One or more Penrose drains are passed into the primary incision and out through the counter drainage incision and fastened.

5. "Posterior levator space" abscesses should never be drained through the rectum, as a "true anal-rectal" fistula may result.

CONCLUSIONS

Operations designed to cure "posterior levator space" abscesses should be definitely considered under the category of major surgery. Since an accurate knowledge of the pelvic diaphragm is of major importance to the success of these operations, this detailed description of a fourth, deep perirectal space, heretofore undescribed, should prove invaluable in anorectal surgery.

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Editorial

PRESENT STATUS OF FEMORAL NECK FRACTURES

THIS fracture still continues to be the "bête noire" of all who treat fractures. The old age group in which the majority of these fractures occur and the mechanical and circulatory complexities of the head and neck of the femur present practical difficulties. In addition, the late complications of aseptic necrosis of the head or hypertrophic arthritis of the hip joint occurring both with and without bony union are becoming more widely appreciated.

The weight of the body at the hip is not borne by direct weight thrust through the long axis of the bone but obliquely through the head, neck and the shaft. This means that longitudinal or lateral traction can not be effectively utilized and, therefore, external or internal splinting must be employed. A long period without weight-bearing is always necessary while this fracture is healing if one is to avoid introducing the added factor of shearing stress and strain.

It has now been about sixty years since Whitman, in 1890, after careful anatomic study of the mechanics of the hip joint announced his abduction method of reduction with external splinting, using plaster of Paris. His method consisted of manual traction, internal rotation and abduction,

all done with meticulous attention to details. Whitman frequently quoted Sir Astley Cooper as representative of the opinion of his day toward this fracture. In 1823 Cooper had stated that the failure of union in this fracture was inevitable because it was impossible to fix the fragments in apposition and he believed that this fact should be officially recognized. He stated that, "Otherwise dissatisfied patients might seek legal redress for a disability for which the surgeon was not responsible." Such a dominant personality as Whitman aroused bitter personal antagonism in his fight against the widespread lethargy in this type of thinking. It must be acknowledged, however, that he certainly succeeded in awakening the profession to a new concept regarding the fundamental principles that were employed in reducing a fracture of the neck of the femur. Today he can truly be considered as "an exponent of a radical reform in this type of fracture."

For many years there have been sporadic efforts to immobilize these fractures by some form of internal fixation and in Moorehead's book, Lambotte refers to Berenger-Feraud who used metal fixation in 1870. John B. Murphy many years ago recommended internal fixation with nails,

and in 1915 Albee described the bone graft peg for this type of fracture. In 1931 Smith-Petersen introduced his three flanged nail, and in 1932 Johansen described the cannulated flanged nail. Both Smith-Petersen and Johansen have introduced an effective method of internal splinting and have stressed the principles of accurate reduction and adequate immobilization. They have, in addition, introduced the factor of early movement of the hip by a method impossible with the Whitman abduction plaster. Other internal fixation methods have been introduced in succeeding years and various types of screws, bone grafts and pins have been employed, the most recent being the spring type of screw fixation which aims at constantly coapting the fractured surfaces. Today an adequate reduction, checked by anteroposterior and lateral views of the hip must be obtained if the patient is to be given the best opportunity for bony union.

Certain important facts regarding the circulation of the femoral head and neck have been worked out by Wolcott, Kolodny, Phemister and others. Wolcott has proven that the ligamentum teres fails to supply the head of the femur in about 20 per cent of adults and that the bone is nourished mainly through the superior and inferior capsular arteries and small branches from the nutrient artery of the femur. Vascularity diminishes as we approach the femoral head and in many hip fractures these blood vessels are severely damaged, making it likely that a certain percentage of cases, irrespective of the methods of fixation employed, will probably go on to non-union. Unfortunately, as yet we have no accurate method to determine the type of intracapsular fracture that one may feel certain will go to non-union. If this could be determined, much time and sometimes life could be saved by immediately resorting to an osteotomy or reconstruction operation, procedures that are today largely reserved for cases with frank non-union.

There are certain mechanical factors that influence healing, one being the valgus position. This position of the head fragment, as well as placing the pin very obliquely so that one flange of the nail engages the heavy cortical bone on the inferior surface of the neck and assures more secure fixation, offers a better opportunity for bony union.

With increasing knowledge regarding geriatrics, a decrease in the mortality rate of the elderly patients has resulted. A committee from the American Academy of Orthopedic Surgeons in 1941 reviewed 923 cases of central fractures, finding a mortality of 11.6 per cent and Boyd has recently reported on 300 patients with this type of fracture and a mortality rate of 9.3 per cent.

In addition, there are local complications which are very serious and which remain largely unsolved. Patients with fractures which unite or remain ununited may develop an aseptic necrosis of the head of the femur or an unusual degree of hypertrophic arthritis. These two complications produce serious results and one finds it difficult to explain how they develop two, three or more years after evidence of solid bony union at the site of the neck fracture. From the best statistics available today one may anticipate the development of late aseptic necrosis in approximately one-third of the cases united by solid bony union! Until these two serious complications have been solved the fracture will still remain what Kellogg Speed has called it, "The Unsolved Fracture."

PAUL C. COLONNA, M.D.

Original Articles

OBSERVATIONS OF BURN SCARS SUSTAINED BY ATOMIC BOMB SURVIVORS

A PRELIMINARY STUDY

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MANY of the burns sustained by atomic bomb survivors have healed with accumulations of excessive amounts of elevated scar tissue, many having the gross appearance of severe scar keloids. An effort has been made, therefore, to collect data and information which might help explain why this occurred. An observational study was made on survivors at Hiroshima and Nagasaki as well as on patients in Tokyo hospitals who had been burned from other causes than the atomic bomb explosion. The work was done jointly with Japanese investigators including physicians in the Japanese Red Cross and Post Office Hospitals in Hiroshima, the Nagasaki Medical College, and the Tokyo Imperial University School of Medicine, the First and Second National Hospitals in Tokyo. This is not intended to be an exhaustive study of the problem.

The burns as seen in 1945 after the atomic bomb explosions were largely flash burns, the result of radiant heat emitted at the time of the explosions and were thus limited to exposed areas of the skin ("profile burns") or to places where overlying clothing was in intimate contact with the skin (especially clothing of dark color).[1] All degrees of severity of burns were seen. In survivors a majority of burns were first or second degree severity, these usually healing rapidly and within four weeks. However, nearly all burns of second degree or worse became infected, this often increasing the severity of the lesions. This was especially true in those individuals suffering from the effects of ionizing radiation, healing usually being delayed in such cases. Thus, many cases of the more severe burns did not heal until October and November, 1945, or later. It was then in these months, after or concomitant with surface healing of these burns, that keloid and hypertrophic scar formation began to be noticed. Treatment of the burns was limited largely to local applications and dressings; relatively few received transfusions of small amounts of blood. Early skin grafting was not done.

The elevations of scar keloids and hypertrophic scars continued, in general, to increase for a few months after their presence was first noticed; a very few were said to be enlarging when seen over one year after the injury. At first, they were red in color, the color gradually fading and after ten to twelve months becoming more brown in color, especially in lesions on the hands. Their surfaces were smooth and shiny, but wrinkling of the surfaces appeared after about a year, indicating contraction. Some have decreased slightly in elevation since the spring of 1946. Many of the individuals with scar keloids complained of hyperesthesias and parenthesias

FIG. 1. Relatively severe scar keloid formation, as seen February, 1947. The patient, age fifteen, was 1.7 kilometer from the ground center at Hiroshima, standing in line with other schoolboys along a street. He wore a khaki blouse which protected him up to his neck. No symptoms or signs of radiation illness were elicited by his history. The keloid had continued slowly to enlarge in elevation. It was brown-red in color and nearly 1 cm. in elevation. The remainder of the left side of his face and neck was hyperpigmented. (Photo by U. S. Army Signal Corps.)

FIG. 2. Scar keloid formation of moderate severity, as seen January, 1947. The patient, age seventeen, was 1.6 kilometer from the ground center at Hiroshima, standing outdoors. No signs or symptoms of radiation injury were noted. The scar was pink in color and was relatively pliable. Most of the surrounding skin of the left cheek was hyperpigmented. This case is typical of the majority of those seen. (Photo by U. S. Army Signal Corps.)

in the lesions, but these symptoms have decreased in intensity with the elapse of time.

The gross appearance of the scarred areas as seen now in large numbers of survivors varies greatly. Some are composed of rather thin atrophic scar tissue while in many other cases the scar tissue is extremely thick and elevated above the surface of the surrounding skin. Vascularity as inferred from the gross appearance also varies a great deal. Judging from gross examination, nearly all burned areas are now completely healed. At the time of this study (December, 1946, to April, 1947) ulcerated areas were still seen in some burn scars and scar keloids, ulceration usually being located at points of stress as in contractures. Large numbers of contractures have occurred at locations where these usually develop. Pigmentary changes are still evident, but the intensity of hyperpigmentation present in many of the exposed skin surfaces has decreased greatly. Hyperpigmentation and depigmentation now serve to outline the original area of burn in many cases. In the central part of some scars, especially those located on the hands and feet, are completely depigmented areas.

The difficulties involved in arriving at a working definition of a scar keloid and the empirical criteria used in this study are

FIG. 3. Relatively severe scar keloid formation, as seen April, 1947. The patient, age twenty, was 1,330 meters from the ground center at Hiroshima, walking along a street in the open. He was wearing a white shirt with sleeves down to his elbows. A partial diffuse epilation of his scalp hair occurred, and diarrhea and gingival bleeding were noted after the bombing. The lower 3 to 4 inches of the keloid on the right forearm were excised July 12, 1946, and the wound then closed. Keloid formation appeared in the line of closure in September, 1946, and by November, 1946, had reached the same elevation that it had been prior to excision. The keloids on the forearms were elevated 1 cm. or more, were firm and their edges were sharply demarcated; at some places they tended to overhang onto the surrounding skin. Each keloid was surrounded by a depigmented area and this then was surrounded by a thin hyperpigmented border. (Photo by U. S. Army Signal Corps.)

FIG. 4. Severe scar keloid formation, as seen February, 1947. The patient, age thirty-six, was 1.5 kilometer from the ground center at Hiroshima, standing with his back facing the center. He sustained flash burns under the half-sleeved white shirt that he was wearing. Epilation of about half of his scalp hair occurred, his hair all returning in October, 1945. Dilated blood vessels were seen on the surfaces of the keloids, especially at their margins. Keloids were elevated about 1 cm. above the surrounding skin surfaces. He complained of pruritus in the keloids, especially in cold weather. The keloids were surrounded by depigmented areas which were then surrounded by a thin hyperpigmented margin peripherally. (Photo by U. S. Army Signal Corps.)

discussed herein. The areas of scar keloid formation as seen now typically occupy the central part of the burned area (where the burn was most severe, probably third degree, and where it healed last) and are surrounded by a depigmented corrugated non-elevated area (this probably having been a second degree injury), this in turn being surrounded at its periphery by a thin hyperpigmented margin. Elevation of keloidal areas varies from a few millimeters to about $1\frac{1}{2}$ cm. above the surrounding skin. Most of the lesions have well demarcated edges. A number of the lesions had claw-like extensions, this feature usually seen along the neck or side of the face.

FIG. 5. Severe sear keloids in a patient who also developed sear keloids in a donor site for a skin graft, as seen December, 1946. The patient, age seventeen, stated that he was located 2.5 kilometers from the ground center at Nagasaki. He was standing in the open, leaning on a shovel with his arms behind him, his back facing the center of the explosion. He wore only short black wool pants and no shirt. Keloids on his arms and back were elevated about 1 cm., were pink in color and were surrounded by a depigmented zone. It was said that a Thiersch graft was removed from his left thigh and applied to his left foot after excision of a sear keloid. Sear keloid formation has developed at this donor site on his left thigh. (Photo by U. S. Army Signal Corps.)

FIG. 6. Marked sear keloid formation at sites of lacerations from shattering glass, as seen April, 1947. The patient, age thirty-eight, was 1.2 kilometer from the ground center at Hiroshima, sleeping in a prone position in the center of a room on the second floor of her home (wood construction). Glass struck her from all directions piercing her clothing and causing lacerations of her back, neck, shoulders and arms. Slight epilation of the frontal part of her scalp occurred in November, 1945, all hair later returning by February, 1946. No burns were sustained. She has had no previous comparable skin lesions. No one else in her family was known to have developed keloids. (Photo by U. S. Army Signal Corps.)

Areas involved vary from a few centimeters diameter to almost complete coverage of the back or forearm. Dilated blood vessels are visible on the surfaces of some scar keloids.

ANALYSIS OF CLINICAL DATA

A major problem in this study consisted of attempting to determine what a scar keloid actually is. It has been found impossible from clinical observations and histologic studies of excised scar tissue to arrive at a working definition that will differentiate scar keloids and hypertrophic scars. If one desires to use both of these terms, the differentiation must be on an arbitrary and empirical basis. It seems regrettable that the word keloid, at least as it has been used up to now, tends to give one the impression that it names a well defined lesion. It would seem that scar keloids and hypertrophic scars differ only in the degree of accumulation of scar tissue after healing of the surface of the lesion. Statistical data on this subject are highly colored by what each investigator's own impression was concerning the clinical appearance of a scar keloid. In general, most investigators tended to consider any elevated scar as a scar keloid.

This study is based on data collected from December, 1946, to April, 1947, on ninety atomic bomb survivors in Hiroshima and Nagasaki. Eighty-seven had been burned while three had sustained lacerations only at the time of the explosions. In addition, twenty-five Japanese patients who were burned by other causes than the atomic bomb explosion were studied in Tokyo hospitals.

Of the total of ninety cases of atomic bomb survivors, forty-nine (54.4 per cent) were considered to have burn scar keloids. The criteria for this clinical classification were purely empirical, a scar keloid being considered as a definitely elevated (3*mm. or more) scar with sharply demarcated borders, the scar usually appearing as a single, rather uniformly elevated mass. This group of forty-nine cases then could be further broken down depending upon the mass and elevation of the scar tissue into severe cases, eight (8.9 per cent); moderately severe cases, twenty-one (23.3 per cent); and mild cases, twenty (22.2 per cent). This use of the term, scar keloid, and this classification serve only to give one an idea of the distribution of the cases as to severity of the lesions. (Figs. 1 to 4.)

Over half of the patients seen were hospitalized for plastic surgery or were outpatients of hospitals and, therefore, included the more severely injured. To obtain a better cross-section view of healed burns of all severity, forty-one of the ninety cases were studied by random selection, i.e., were from groups of school children and post office employees in which every burned person was studied. Of these forty-one, nineteen (46.5 per cent) were considered as showing scar keloids.

In the Tokyo hospitals twenty-five patients being treated for scars which resulted from burns of other causes were seen. Nearly all cases had been burned over one year before, eighteen out of the twenty-five having received their injury between eighteen and twenty-four months previously. This was a relatively select group in that they included the more severely burned

FIG. 7. Severe scar keloids following healing of flash burns due to a gasoline explosion, as seen February, 1947. This boy, age nineteen, sustained burns April 13, 1945, in Tokyo, when gasoline exploded nearby, secondary to the explosion of an incendiary bomb. These lesions are entirely comparable to those seen in atomic bomb survivors. The burns of this patient occurred only on exposed skin areas, as shown by the sharp lower edge of the keloid on his neck, below which a collar protected the skin. The scar keloid was pink in color. (Photo by U. S. Army Signal Corps.)

cases, namely, those requiring hospitalization for treatment of extensive scars. Twenty were considered to have sustained flash burns from incendiary bomb or gasoline explosions, four were burned by flames or after their clothing ignited, one by a gunpowder explosion and one by nitric acid. Of the twenty-five patients, there were ten (40 per cent) considered to have developed scar keloids. (Fig. 7.)

The patients in the Tokyo hospitals had apparently received more care than atomic bomb survivors during the period just after the occurrence of their burns; but the type of treatment was much the same as that given to the atomic bomb survivors, consisting largely of local application of oint-

ments with dressings. Six had been grafted but none was grafted within four months after his initial injury and in most cases grafts were applied after six months. The skin grafting was done when the burns were completely healed or nearly healed with scar tissue.

It was impossible to judge at the time of this investigation what the severity of the burns from any of the groups had been initially, especially in view of the fact that infection, as was universally present in burns of atomic bomb survivors, could have increased their severity as to depth and area involved. Surfaces of all of the scar keloid areas were thought to present the appearance of scars following the healing of third degree burns. Thirteen of the atomic bomb survivors who were classified as not possessing scar keloids did have scars thought to present the appearance of scars following healing of third degree burns.

There were four cases of scar keloid formation at the sites of lacerations which resulted from shattering glass at the time of the explosion. (Fig. 6.) Keloid formation in such lesions actually seemed to occur only infrequently. In a random selection of twenty-six other individuals (nurses and post office employees) who had scars of lacerations, none had developed scar keloids.

Most of the cases of scar keloids (64 per cent) and also patients considered to have only hypertrophic or usual scars (75 per cent) were between the ages of eleven and twenty. Literature on the subject indicates that keloids do occur most frequently in this age group, but the data accumulated in this study are from selected groups and are insufficient to permit a definite conclusion.[2,3] Of the ten cases of scar keloids in the control series, one was in the one- to ten-year age group, two were between eleven and twenty, three were between twenty-one and thirty and three were between thirty-one and forty. Eleven of the fifteen remaining patients with ordinary type scars were between twenty-one and thirty years of age.

Most of the survivors studied were located between 1,500 and 2,000 meters from the ground center at the time of the atomic bomb explosions. This is understandable since, in survivors, the "burns were greatest not only in incidence but also in severity at about 1,500 to 2,000 meters."[1] The majority of those who had been closer to the center and so located as to sustain a severe burn did not survive. Approximately one-third of both the group of scar keloid patients and the group with ordinary type scars were within 1,500 meters of the ground center. In the series of scar keloid cases, only one had been beyond 2,500 meters of the ground center, this case having been located at 2,900 meters. There was no consistent relationship between distance from ground center of the explosion and the incidence of scar keloid formation except that which seemed well correlated with the variation of severity of the lesions with distance.

It was believed that plastic surgery had been done on too few of the patients to warrant conclusions regarding the frequency of recurrence of keloids and occurrence of keloids in donor sites for skin grafts. A number of patients had recently undergone surgery, but the final outcome is yet unknown. Three of ten patients who had been grafted following removal of scar keloids showed recurrence of keloids, and two others developed hypertrophic scars in grafted areas. These recurrences were especially located where grafts had apparently not taken well. Four of eight patients developed keloids again along lines of closure following excision of keloids with primary closure. Three of eight patients showed development of keloids in donor sites, two said to have been Thiersch grafts (although the type of scar now present after keloid formation would indicate that they might have been deeper) and one at the site of "pinch" grafts. (Fig. 5.)

In the group of patients in Tokyo hospitals, one of six cases showed recurrence of

keloids in grafted areas. Scar keloid excision with primary closure was done in one patient without recurrence of the keloid. Two of four cases were thought to have developed very mild scar keloids in donor sites for full-thickness grafts.

burns sustained from the atomic bomb explosion, but only three other persons stated that other surviving members of their family had been burned. Two of the patients showing scar keloid formation at the sites of lacerations are mother and

<div align="center">TABLE 1</div>
<div align="center">INCIDENCE OF SCAR KELOIDS IN SCHOOL CHILDREN</div>

Group	Distance from Ground Center	Total No. Seen	No. Burned	Patients with Keloids		No. Having Radiation Illness
				No.	Per Cent	
First High School, Hiroshima Prefecture.......	1.6 km.	75	46	41	(89.1)	9
Second High School, Hiroshima Prefecture.....	2.1 km.	71	55	52	(94.5)	5
Commercial School, Hiroshima Prefecture.....	2.1 km.	130	114	95	(83.3)	4
Second High School, Hiroshima Prefecture.....	2.3 km.	159	156	51	(32.6)	3
First High School, Hiroshima Prefecture*......	1.2 km.	16	1	1		16
Third Primary School, Hiroshima Prefecture...	1.3 km.	24	19	7	(36.8)	24

* A total of 150 persons were in this group; about one-half died as a result of mechanical injuries and radiation injury. This group was indoors; only one person sustained burns.

The anatomic distribution of scar keloids depended, of course, upon the distribution of the burns and, therefore, were seen largely on exposed skin surfaces. The head and face were involved in 21 per cent of cases, neck in 24 per cent, arms in 12 percent, back in 10 per cent, forearms in 9 per cent, anterior chest in 6 per cent, legs in 6 per cent, shoulders in 5 per cent, dorsum of hands in 5 per cent and feet in 2 per cent. In the Tokyo group of control patients the head and face were involved in 26 per cent, dorsum of hands in 20 per cent, neck in 16 per cent, forearms in 16 per cent, legs in 11 per cent and feet in 11 per cent. Eight of twenty-five atomic bomb survivors who were burned beneath clothing developed scar keloids at the sites of these burns. Of the Tokyo patients, three sustained burns beneath clothing but none developed scar keloids in such burns.

Six of the atomic bomb survivors stated that their keloids were still increasing in elevation. Five still complained of chronic ulcers at sites of scar keloids. Many had developed contractures. Five patients gave a history of another member of their family having developed scar keloids in

daughter; they were together at the time of the explosion. No case studied had ever developed scar keloids at the site of any skin lesion prior to the atomic bomb injury.

There were thirty-one patients giving a history of signs or symptoms of radiation illness (epilation, diarrhea, hemorrhagic tendency) after the bombing. There were four cases with a questionable history. All of these individuals were within 1,800 meters of the ground center at the time of the explosion. Twenty-two of these thirty-five patients were considered to have developed scar keloids; twelve did not.

Reports by other investigators all give what appear to be relatively high figures for the incidence of keloids in burn scars in atomic bomb survivors. The figure will vary with the investigator, his concept of the clinical appearance of a scar keloid and the group of survivors studied. Hatano, Uchida and Watanuki from the Tokyo Imperial University School of Medicine surveyed groups of school children at Hiroshima in December, 1946.[4] Persons in each group were at approximately the same location at the time of the explosion. All patients who sustained burns were out-

side in the open. The results of their study are shown in Table 1.

Dr. Yoshio Ito, dermatologist at the Hiroshima Japanese Red Cross Hospital, in a ·study of 606 burn cases between September, 1945, and February, 1946, found 210 (34.7 per cent) that he considered to have developed scar keloids.[5] He emphasizes that this incidence is that only of scar keloid formation, those cases with hypertrophic scars having been carefully excluded. These scar keloids appeared from one to six months after the bombing, with 100 out of the 210 appearing during the second and third months.

In a study of 501 highly selected survivors reporting to out-patient clinics of Hiroshima hospitals in November and December, 1946, Lt. (jg.) Fred M. Snell (MC) USNR found that 258 had sustained burns, and, of these, 203 (78.7 per cent) showed scar keloid formation in varying degrees.[6] In addition, there were nineteen patients who had developed scar keloids secondary to injuries other than burns.

MICROSCOPIC OBSERVATIONS

A total of eighty-one specimens of lesions considered clinically to be scar keloids removed from atomic bomb survivors from March, 1946 to April, 1947 were studied histologically. Also, sections from biopsies from edges of healing burns obtained in October and November, 1945 were examined. Three sections from lesions considered as ordinary burn scars on atomic bomb survivors, three sections of similar lesions on Japanese burned from other causes and four incisional scars (three of them incisional scars following sequestrectomy in osteomyelitis) were studied. Sections from one spontaneous keloid and eleven sections of scar keloids which developed in Japanese burned from other causes than the atomic bomb explosion were also seen.*

Microscopically, it was found impossible to reach definite conclusions as to the specific configuration of keloids. It seemed impossible to distinguish what might be considered as hypertrophic scars from scar keloids, or from any large mass of pure scar tissue, for that matter. Comparison of all specimens showed much variation in details of morphology, distribution and abundance of all structures, the clinical types of lesions being distinguishable, for the most part, only by the quantity of fibrous connective tissue present. There was no evident consistent difference between the spontaneous keloid, scar keloids developing in healed burns of atomic bomb survivors and scar keloids of healed burns of other etiology. It is said that in the early stage of true or spontaneous keloids, normal tissue is seen in the papillary layer of the dermis above the "tumor mass." In both types of keloid formation the growing mass of tissue is said to be formed in what may be considered as the lower strata or reticular layer of the dermis.[7] However, from this present study one can only agree with Heidingsfeld who concluded in 1909 that after a review by differential histopathology of the character of the location of the ·tissue of the lesion, preservation or absence of overlying papillae, elastic fibers, hair follicles and glandular structures, the arrangement and distribution of the fibrous tissue, vascular changes and relationship to blood vessels, it is impossible to distinguish idiopathic keloids, scar keloids, hypertrophic scar or ordinary scar tissue.[8] (Figs. 8 to 12.)

The scar keloids were seen to be composed of massive quantities of interlacing collagenous connective tissue fibers, between which were connective tissue cells varying in concentration with the specimen. In general, the connective tissue cells were relatively numerous, there being a gradual decrease with age of the specimen, but this was not consistently true. The cells in many cases appeared long and oval in shape with large vesicular nuclei. In other cases the cells were more spindle-shaped and smaller in size. No conclusions could be reached as to the presence of any

* The U. S. Army 406th General Medical Laboratory in Tokyo very kindly prepared these sections for microscopic study.
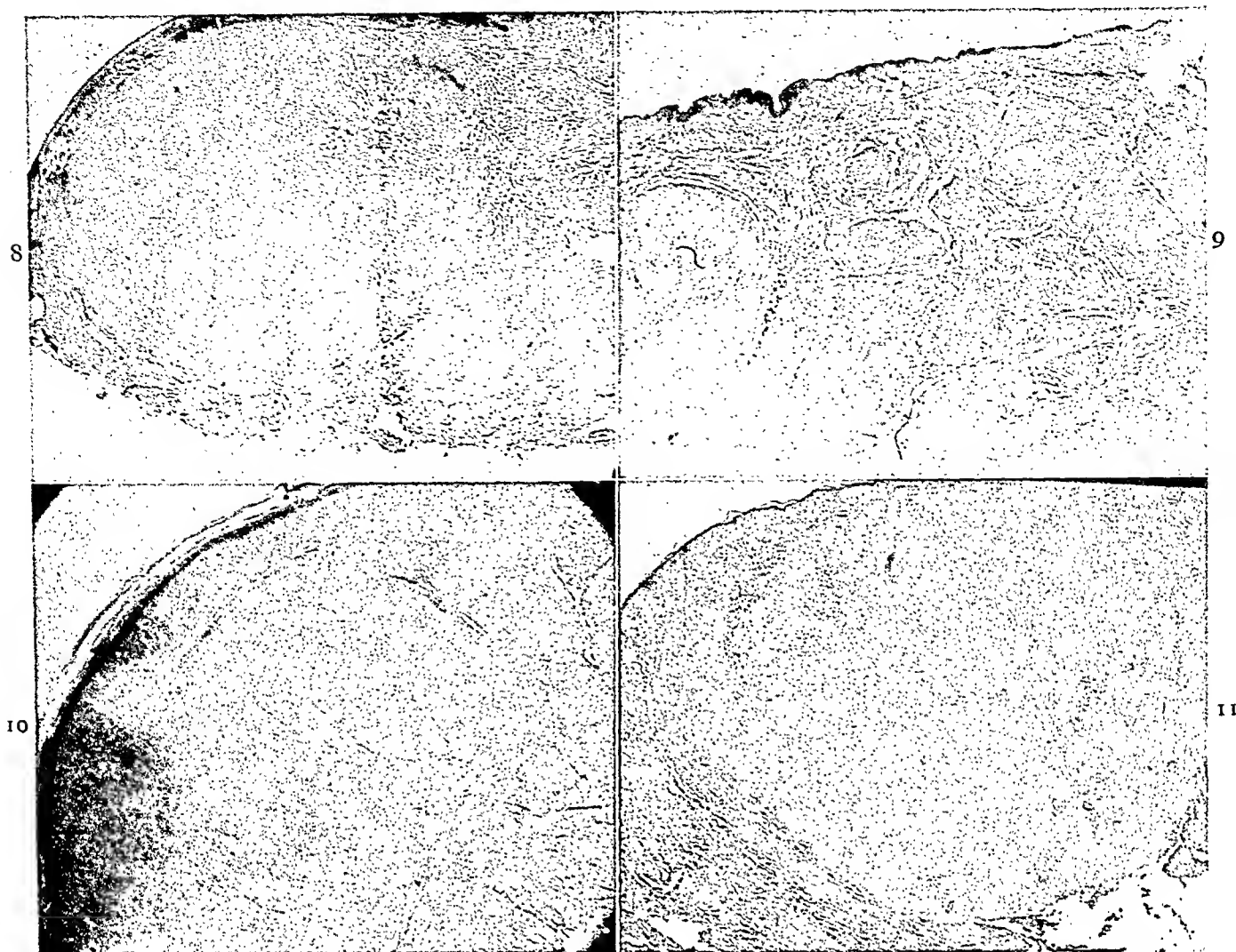
FIG. 8. This is a section from a scar keloid removed January 21, 1947, from the neck of a fifteen year old atomic bomb survivor of Hiroshima. This section is taken from the edge of the lesion, the edge being shown on the left side above. It is composed largely of a large dense mass of collagenous fibers. This demonstrates the manner in which this mass compresses slightly the narrow layer of tissue between it and the epidermis above and compresses and slightly bulges into normal appearing tissue at the edge of the lesion (shown at the left side of the illustration). The mass also seems to be distinct from a thin layer of tissue separating it from the subcutis. In this latter thin layer are seen sweat glands. The overlying epithelium is thin and lacks rete pegs. This illustrates a pattern frequently seen in the sections of scar keloids studied; hematoxylin and eosin, X 14½. (Neg. No. 101064.)

FIG. 9. This is a section from a scar keloid removed from the dorsum of the hand of a 20 year old Hiroshima atomic bomb survivor on 30 January, 1947. This section illustrates another pattern seen in the scar keloids studied. It is composed of collagenous fibers arranged as a number of large relatively dense bundles, between which are running interlacing collagenous fibers in a more loose arrangement. The epithelium is thin and its basal cells are heavily pigmented; hematoxylin and eosin, X 16. (Neg. No. 101065.)

FIG. 10. This is a section from a scar keloid removed July 30, 1946, from a thirty-four year old Hiroshima atomic bomb survivor. This illustrates another pattern seen in the sections studied. The epithelium is thin and lacks rete pegs. The underlying tissue is rather homogeneous in appearance consisting of a large amount of collagenous connective tissue fibers running, for the most part, parallel to the epithelial surface; hematoxylin and eosin, X 18½. (Neg. No. 101077.)

FIG. 11. This section was taken from a lesion considered to be a scar keloid which developed on the chest of a Japanese patient, age twenty-three, burned by an incendiary bomb explosion, not by the atomic bomb explosion. The injury occurred July 23, 1945; the lesion was excised July 4, 1946. This illustration shows an area at the edge of the lesion, the junction with normal appearing skin being seen on the left. The epithelium is not unusual. Large masses of dense collagenous tissue fibers are located in the deeper part of the dermis compressing slightly the adjacent tissue. At the base of the section, just above the subcutis, and in the normal cutis adjacent to the mass are normal skin appendages; hematoxylin and eosin, X 160. (Neg. No. 101076.) (Figures 8 to 11 were made by the Army Institute of Pathology.)

Fig. 12. × 23 magnification. H and E. This is a section from a lesion considered to have been a spontaneous keloid that developed on the anterior chest of a Japanese patient, age thirty-seven. The keloid was said to have been noticed first eight years prior to surgical removal which was done July 12, 1944. This illustration shows an area at the edge of the lesion, the junction with normal appearing skin being seen on the left. The epithelium was not unusual. The mass of tissue was composed of some large collagenous fibers with some bundles of finer fibers more densely packed. (Neg. No. 101075). (Photomicrograph by Army Institute of Pathology.)

specific pattern or configuration of the connective tissue in the various types of lesions. Many appeared as single or several large dense masses composed of interlacing bundles of thick collagenous fibers, these masses occupying what might be considered as the reticular layer of the dermis and bulging into or slightly compressing the thin layer of subepithelial fibrous tissue above (in what might be considered as the papillary layer of the dermis), the normal and scar tissue at the edges of the lesion, and also the normal and scar tissue in or near the subcutis at the base of the lesion. (Figs. 8, 11 and 12.) In other specimens the large quantity of scar tissue was composed of many bundles of dense interlacing thick collagenous fibers running in various directions and embedded in fibrous tissue arranged more loosely. (Fig. 9.) Still another pattern seen in some specimens was that of a homogeneous distribution of interlacing collagenous tissue fibers traveling, in general, parallel to the epidermal surface. (Fig. 10.)

No sections were seen in which elastic tissue was present in the fibrous tissue. Elastic tissue occasionally was seen in tissue just above the subcutis and just below the fibrous tissue mass. In some sections the enlarging growth of connective tissue had apparently enveloped some sweat glands, sweat ducts and occasionally a hair shaft, such structures usually being seen near the edge of the lesion and near adjacent normal dermis. Normal skin appendages as sweat glands were frequently seen in tissue just at the level of the subcutis and just below the fibrous tissue mass. Sweat ducts were seen running through the scar tissue in some of the nonelevated or slightly elevated scars. Vascularity of the specimens varied but, in general, the tissue was not very vascular. Metachromatic staining and early hyalinization of the collagenous fibers were seen in some cases studied.

The epidermis overlying this scar tissue varied in appearance, being most often thin and lacking rete pegs. These latter changes had no constant relationship with the appearance of the underlying connective tissue. The basal cells of the epidermis appeared to contain excessive quantities of pigment in many cases although in other cases they appeared to be completely devoid of it.

Small collections of inflammatory cells were seen in some sections. These included lymphocytes, macrophages, and plasma cells and were usually found as a collar around blood vessels near the periphery of the expanding growth of collagenous fibers or in adjacent normal or scar tissue. Such cells were found occasionally around sweat glands or as independent groups in a small area of tissue or fat necrosis. Foreign body giant cells were seen in several sections around a foreign body or in an area of apparent fat necrosis. It was thought impossible to arrive at any conclusion as to the significance of these findings, as to whether they had a cause or effect relationship or were unrelated to the scar keloid and hypertrophic scar formation.

Sections of forty-one biopsies of very small amounts of tissue taken from the edges of healing burns of atomic bomb survivors at Hiroshima approximately three months after the injury were all found to be composed of granulation or scar tissue homogeneously arranged beneath the epithelium. In twenty-six of the sections there was some evidence of an inflammatory process as indicated by perivascular collections of small focal, or occasionally, diffuse collections of macrophages, lymphocytes and plasma cells. Again, it was deemed impossible to fix any relationship between this finding and scar keloid or hypertrophic scar formation. The final outcome of the healing of the lesions of this group of cases is unknown.

COMMENTS

A high incidence of hypertrophic scars and scar keloids did occur in Japanese survivors of the atomic bombings. What is the significance and explanation of this?

A review of the literature on the subject leaves one confused as to what a scar keloid actually is, what the features which specifically distinguish it clinically and histologically from ordinary scars or hypertrophic scars are and what the etiology and pathogenesis of such a formation is. Some state that scar keloids may be differentiated from hypertrophic scars by the fact that scar keloids extend laterally beyond the limits of the original lesion while hypertrophic scars do not.[7] The clinical and histologic studies presented above indicate that the difference between non-elevated scars, hypertrophic scars and scar keloids is only one of degree of accumulation of fibrous connective tissue.

There are a number of pieces of information which tend to preclude the attachment of any special significance to scar keloid formation in atomic bomb survivors. Scar keloid formation was seen in Japanese burned from other causes than the atomic bomb. Although the incidence of spectacular large scar keloids in such patients did not seem as high as in atomic bomb sur-

vivors, some cases were seen that would about equal in severity the most severe cases of atomic bomb survivors. (Fig. 7.)

Hiraga states that according to the hospital records of the Tokyo First National Hospital, 106 patients with recent or old burns have been seen as out-patients or were hospitalized from December, 1946 through March, 1947.[9] Of these, twenty-three were considered as having developed scar keloids while seventy-two were thought to have developed hypertrophic scars. In addition, five atomic bomb survivors who had sustained burns were seen, one of whom was thought to have developed scar keloids.

One of the first questions presenting itself is whether the Japanese people have a predisposition for keloid formation. In general, available information indicates that perhaps they have a slightly greater tendency for keloid formation than the white race, but not so great a tendency as the negro race. Hiruma presents the best information on this subject.[3] In a study of 82,950 patients who came to the Tokyo Imperial University, Dermatology Department, with all types of skin diseases during the years 1930 to 1939, 296 complained of keloids. This gives a ratio of 7.8 per 2,000. In a review of Japanese literature on the subject, he found the highest figure of 8.7 per 2,000 given by Yamazaki and the lowest figure of 2.9 per 2,000 given by Ogata, Otani, and Hata. These figures then may be compared with those of Hazen who found a ratio of one case of keloid in 2,000 white patients and fourteen (later twelve) in an equal number of negroes.[10,11] Fox found three cases of keloid in 8,382 white patients and seventy-six in 11,486 negroes, a ratio of about nineteen negroes to one white person.[12] Matas reported a ratio of negroes to white persons of nine to one.[13] It is to be noted that these figures refer to keloid arising in all types of scars or spontaneously, and the incidence of such keloids is based on groups of individuals with all types of skin lesions. Actually, the many possible variables inherent in studies such

as these make it difficult to compare their results. No statistics could be found as to the general incidence in Japanese people of scar keloid formation in burn scars or the incidence of keloid formation in third degree burns.

Hiruma gives the type of lesion in which the keloids developed in 230 of his cases.[3] Injuries were given as the cause in 120 cases: burns, twenty; surgical operations, twenty-nine; wounds, ten; scratches, four; corrosions, seventeen; and others, ten. There were sixty-eight cases in which a suppurative process (as furuncles) was the original cause. The origin of thirty-eight cases was in lesions of other skin diseases.

Of course, scar keloids do occur in the white race and the term is frequently found in references to the types of scars resulting in untreated severe burns. It is especially interesting to note that in the report of survivors of burns from the Cocoanut Grove disaster treated at the Massachusetts General Hospital, it was found that all patients (seven) with third degree burns not grafted later developed what are called keloids.[14] No patients with third degree burns who were grafted developed keloids. Keloid formation probably was not striking in severity in these cases but at least definitely elevated scar tissue apparently did occur.

It is well known that if third degree burns are permitted to heal independently and are not grafted early, elevated scar tissue or what are called scar keloids do frequently occur.[15,16,17] This is especially prone to occur in cases presenting delayed healing and serious infection of the tissue of repair. It is only during the last few years that a deeper appreciation of the special significance of full-thickness burns has arisen. Cope et al. stress the importance of grafting these burns as soon as possible after the occurrence of the injury and the advisability of grafting upon the tissue at the base of the excised necrotic tissue, not grafting upon granulation tissue.[18] They illustrate a case in which one leg was covered on the seventh day with sheet grafts after surgical excision of the slough of the third degree burn. The other leg was grafted on the forty-seventh day with postage stamp grafts on the granulation tissue. After three months it was noted that keloidal tissue had formed diffusely beneath the grafts on granulation tissue whereas no keloid scarring occurred at the location of the sheet grafts except along suture lines. One gains the impression that about all of the hypertrophic scars and scar keloids of atomic bomb survivors occurred in what were deep second or third degree injuries initially or later as a result of infection.

The burns in the survivors were practically all flash burns. Work of Moritz indicates that such burns when extending into the dermis result in a coagulation necrosis of the collagen which heals slowly because of the resistance of the denatured collagen to autolysis and organization.[53] He too notes that the superficial appearance of a burn is not a reliable criterion in judging the depth of a burn and suggests early biopsy of a burn to obtain such information to aid in the proper early treatment.

The burns sustained by atomic bomb survivors certainly healed under conditions excellent for the development of hypertrophic scars or scar keloids. One certainly wonders if scar keloids would have occurred if it would have been possible to graft all full thickness burns soon after the time of the injury. It is said that all burns of second degree or worse became infected and in some cases this resulted in destruction of parts of the skin that had not been injured by the primary burns.[1] The healing of burns are delayed in many cases, with some cases showing incomplete surface healing five months or more after the injury. Some cases also sustained radiation injury which caused a diminished ability to combat infection and to heal and resulted in prolonged healing and increased severity of the wounds. Many cases suffered from marked malnutrition and a poor general condition as a result of the systemic effects of the burns and the lack of food which was

especially pronounced in Japan after the termination of the war. Treatment was minimal and consisted largely of local applications of various greasy ointments and dressings, often by the patients themselves or their relatives. Early skin grafting was not done.

It is impossible to evaluate definitely the significance of the factor of the direct action of ionizing and neutron radiation in the production of these scars. The fact that many patients with scar keloids were located sufficiently far enough away from the center at the time of the explosion so that the amount of ionizing or neutron radiation that they received must have been very low, plus the fact that the burns appeared immediately and were limited for the most part to exposed areas of the skin and were flash burns, tends to minimize the importance of the direct action of these radiations. Any relation between distance from the ground center of the atomic bombings and the frequency of scar keloid formation appeared to be a reflection only of the variation of the severity of the burns. It does seem most probable that any ionizing radiation received by survivors at the time of the bombing was practically all "hard" in character and consisted of gamma rays. Furthermore, it seems that the contribution of residual or long term radioactivity following the immediate explosion in causing or altering the healing of the burns was insignificant.

Why do scar keloids or excessive amounts of scar tissue form? Again, a review of the literature leaves one confused. A number of theories have been advanced, but adequate supporting evidence to prove the etiology or complete pathogenesis of scar keloids is lacking. A number of investigators as Garb and Stone consider hormonal stimulation as being the main or the important cause of keloid formation since the greatest frequency occurs in the second decade of life when an increased glandular stimulation occurs.[2,19] They point out also that keloids are rare in the aged. Geschickter attributes keloid formation to a localized

endocrine disturbance.[19] He found increased gonadotrophic substance and estrin on assay of a keloid from the ear. A number of pieces of circumstantial evidence indicate that there are endocrine influences in the tissue regeneration in skin healing.

Marshall believes that keloid formation depends upon excessive serous exudation in wounds, this exerting a chemotropic response from fibroblasts which migrate to the area and proceed to produce an excessive amount of scar tissue.[20–23] In support of this he has shown that repeated intracutaneous injection of serum result, when studied histologically two or three weeks later, in condensation, increase and thickening of subepithelial collagen. Marshall points out that tissue fluid extravasation which occurs in various diseases is followed by connective tissue formation in those areas. Sheehan has used hygroscopic threads introduced at three levels to drain fluid from keloids and reports the occurrence of blanching, subsidence, and pliability of the scar, the excision of which can be accomplished subsequently without recurrence.[54]

Glücksmann considers the formation of hypertrophic scars and scar keloids as a special type of foreign body reaction occurring in sensitive persons.[24] He studied serial biopsies of these lesions from about seventy patients at intervals of one week to several years after injury. The foreign bodies were considered to be extrinsic, as dust particles, but more often intrinsic, as keratinized cysts or abnormal sweat glands. Such bodies were thought to elicit a cellular reaction and perivascular infiltration of fibrocytes. A cellular tissue first formed around the vessels, later changing into the typical tissue of the scar keloid. Krysztalowicz considers both idiopathic and scar keloids to be the result of a chronic inflammatory process such as chronic inflammation of hair follicles and sweat glands.[28]

In a study of burns, Cope and his associates state that "it is an impression that the keloid and scarring are roughly pro-

portionate to the amount of the pre-existing infection."[18] They point out that although chemotherapy will hold invasive infection in abeyance, infection of full-thickness burns is inevitable if they are not grafted shortly after the injury. Gordon and his associates showed that in third degree burns, in contrast to second degree burns, pathogenic organisms seldom behave as innocent residents.[29] A high incidence of severe infections occurred in burns of atomic bomb survivors. Perhaps it would be a mental satisfaction to consider keloids as the result of repeated attempts at repair, each partly frustrated by new injury, so that an excess of scar tissue finally results, but sufficient evidence to support such a hypothesis is lacking. Keloids occur even in skin wounds healing by primary union.[2]

Other investigators have more specifically fixed the origin of keloids as from cells of the adventitia of blood vessels in the corium.[7,25,26,27] Ormsby refers to Warren as stating that keloids begin "by a growth of round cells in the adventitia of the arterioles of the corium. These cells become fusiform, and finally develop into the connective tissue fibers forming the tumor."[7] Ormsby also states that masses of cells usually surround blood vessels extending from the main mass of the keloid and that it is from these cells that recurrences form after supposed removal of the tissue mass. After a histologic study of thirty keloids, three hypertrophic scars and three cases of scars in Japanese subjects, Hiruma concludes that the cells forming a keloid differentiate from the adventitia of blood vessels.[27] He considers the morphology of these cells as being peculiar and found only in keloids and not in hypertrophic scars. He could find no histologic difference between idiopathic and scar keloids. In some of the sections from atomic bomb survivors and from Japanese burned from other causes the general arrangement of fibers was in concentric rings, these occasionally around blood vessels. No impressions were gained in the present study concerning extensions along the course of adjacent blood vessels. Heidingsfeld could not trace the origin of keloid formation to the adventitia or extensions along blood vessels of the corium. He points out that "the tendency for a keloid to become self-limited in its clinical growth and not to involve the deeper subcutaneous structures, also speaks strongly against a vascular origin and an extension along blood vessels. The involvement of blood vessels seems not to be a primary process but a secondary one due to the direct independent extension of the body of the growth proper. In this way the blood vessels become invaded and involved, in the same manner as the hair follicles, sudoriferous and sebaceous glands and other specialized structures of the skin."[3]

Bohrod presents a hypothesis that keloids are now seen more frequently in negroes as a result of the process of sexual selection. This is based on the assumption that a hereditary physico-chemical difference is present in certain individuals causing a tendency for keloid formation. Since in the cult of the African negroes there apparently arose the belief of a magic association between keloid formation and fertility, with the passage of time individuals with a predisposition for keloid formation formed an increasingly greater portion of their population.

There is evidence that the tendency to form keloids not only varies with races but also that there is in some cases a familial tendency.[31,32,33] A number of reports describe cases with hereditary features, but this has never been sufficient to explain such formation in more than a few individuals. The factor of an individual or familial predisposition certainly is to be considered in some cases but the fundamental characteristic which manifests itself by such a predisposition is unknown.

Other investigators have been able to understand keloid formation only as being the result of the occurrence of biochemical and biophysical alterations in the healing process. To explain recurrence of keloids, some investigators must assume that some

chemical change in the local tissues has occurred.[34] Filips believes that the "underlying cause of keloid formation is the hypersensitivity of the fibroblasts and of the vascular endothelial cells to any agent having the power to mobilize those elements in the protein of these cells which induce them to proliferate."[35] He also observes that the "keloidal tendency of the skin varies directly with the amount of tissue destroyed below the papillary layer of the skin and depth of the destroyed tissue, but at the same time large wounds produced by most of the physical and chemical agents and extending into the reticular layers of the cutis or subcutis are potential keloids, regardless of the keloidal tendency of the skin in which the wounds are produced."

Knowledge concerning the biochemical and biophysical factors and processes in the steps of normal wound healing is scanty. Both in vivo and in vitro studies indicate in early wound healing sequences of, first, active fibroblastic proliferation, followed by a period of relatively slow cellular division during which time the large amounts of collagen are formed.[36,37] What controls the quantity of collagen deposited and the period of time over which this occurs is unknown. The pH of the medium seems to influence the quantity of collagen produced.[37,38] The most favorable pH for collagen deposition is said to be that normally found in the body. Evidence obtained by available methods does indicate that collagen fibrils and fibers are formed extracellularly in the presence of fibroblasts.

Growth stimulating substances and substances stimulating respiration and glycolysis are said to be produced by injured cells according to investigations using ultraviolet radiation to produce controlled tissue injury.[39] Carrel demonstrated a product of leukocytes which excites fibroblastic proliferation and he also observed that extracts of muscle and gland tissue of adult animals stimulate rate of growth of homologous fibroblasts in vitro.[40,41,42] Caspe

found that creatine increased the rate of growth of a variety of tissue cultures, including fibroblasts.[43] Fell and Danielli believe that alkaline phosphatase is connected with the laying down of collagen.[44] Vitamin C and the body protein levels are considered as important factors and substances in wound healing.[45,46] Tissue tension is a mechanical factor to be considered, and should be remembered in severe injuries where the architectural framework of a large area of tissue is destroyed, as in severe burns. The connective tissue of the body as a dynamic physiologic system itself, changing with age, has increasingly become more apparent in recent years.[47] Present work on hyaluronic acid and related substances should appreciably increase our understanding of connective tissue and its formation.

Several studies of the healing of cutaneous wounds in man further corroborate the findings and impressions gained in this study of the Japanese. Converse and Robb-Smith studied 469 skin graft donor areas, the grafts having been of all thicknesses.[48] They concluded that the quality of the repair was roughly proportional to the rapidity of healing, donor areas healing in six to ten days leaving only a faintly visible scar with a soft pliable base, those healing in fourteen to twenty-one days leaving a more visible uneven scar with a harder base, while those remaining unhealed for a longer period of time often left retracted hypertrophic scars. The rapidity of healing in turn depended upon a number of factors. Donor sites for thin Thiersch grafts healed in six to ten days, for intermediate grafts within fourteen days, whereas healing in areas cut along the level of the base of the dermis required twenty-one to fifty-eight days. In infected donor areas the average healing time was fifty-two days and a rough hypertrophic scar remained. Healing occurred faster where the dermis was thicker and where the skin was loosely bound-down to underlying structures. Illustrative photomicrographs very nicely show the difference between the healed

donor sites for thin and thick grafts, the healed hypertrophic scar at the donor site of a thick graft appearing very similar to the scar keloids and hypertrophic scars studied in the Japanese.

Bishop investigated the regeneration of skin after its removal to various depths in man.[49] He found that if the derma was removed down to the reticular layer, granulation tissue regenerates readily from connective tissue about the hair follicles and full thickness regeneration of the derma occurred. However, if the removal extended into the reticular layer and was sufficiently deep to remove the bases of the hair follicles, scar formation, often pronounced, occurred. The skin may be repeatedly removed from the same area and regeneration as an even sheet of new tissue will occur to full thickness with normal texture and without scarring as long as the removal is above the reticular layer. It was found that histologic differentiation into normal or into scar derma takes place only after healing of the surface epithelium, the fibrotic hyperplasia characteristic of scar tissue occurring after this time. Bishop suggests that there is some tissue element present in the papillary layer which regulates normal growth of connective tissue; if the papillary layer is destroyed, granulation tissue undergoes fibrotic hyperplasia and forms scar.

The natural history of scar keloids in the Japanese is as yet incomplete. It is known that neoplastic changes do occur in such lesions, but in man they are virtually all of epithelial origin, occurring only infrequently and usually after many years and especially in those lesions which healed very late or were prone to recurrent ulceration. Treves and Pack state that about 33 per cent of all scars giving rise to carcinoma are produced by burns.[50] According to Hueper, between 9 and 25 per cent of all cases of carcinoma on the extremities develop in scars of burns.[51] Japanese dermatologists state that skin neoplasms, especially carcinoma of the skin of the face, are less frequent in the Japanese than in the white race.[52]

SUMMARY

A relatively high incidence of scar keloid and hypertrophic scar formation of a severe degree has occurred after healing of flash burns that probably were of deep second or third degree severity and in a people (Japanese), some of whom perhaps have a predisposition for the development of scar keloids. At this late date, the lack of complete, detailed, continuous studies and records on a large group of cases dating from the time of the occurrences of the injury and the large number of variables to be considered render impossible a complete evaluation and understanding of scar keloid formation. The inadequate treatment, poor nutrition, high incidence of severe infection and delayed healing should be considered as important contributing factors which affected the healing process to result in a high incidence of severe keloid or excessive scar formation. Scar keloids were found to occur in Japanese sustaining burns from other causes than the atomic bomb explosion. It would seem most probable that the scar keloids represent no peculiar effect of the atomic bomb explosion. Furthermore, it seems probable that a similar incidence of occurrence of scar keloids could have occurred in burns of the same severity from any other cause under similar conditions during the healing of the lesions in patients having the same general state of health.

One gains the impression that the pathogenesis of keloids can be completely explained only by a better understanding of the detailed biophysical and biochemical processes which occur in the healing of skin lesions and how the initial conditions and possible later alterations in these steps influence the final result of the reparative process. Some individuals may be so constituted that they have a tendency to develop excessive amounts of scar tissue in the healing of wounds. The factors involved

in the etiology of keloids are probably multiple.

It is difficult to arrive at a differentiating working definition of scar keloids. It seems most probable that the differences between ordinary non-elevated scars, hypertrophic scars and scar keloids are only those of degree of amount of fibrous connective tissue produced during the healing process. From clinical data, histologic observations of various types of excised scars and reported experimental studies of the regeneration of skin in man, it would appear that the excess collagen production causing scar keloids and hypertrophic scars occurs when the lesion extends deep in the reticular layer of the dermis and, therefore, occurs usually in burns which extend to this depth initially, or later as a result of necrosis caused by infection or additional trauma. The necessity for early grafting of full thickness burns is again demonstrated. The necessity of preparation for the early care of burns in great numbers of casualties in the event of a catastrophe such as an atomic bombing of a populated area is obvious.

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TUMORS OF THE CAROTID BODY*

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BECAUSE tumors of the carotid body are of infrequent occurrence and present unique problems, operation in these cases has resulted in an unnecessarily high morbidity and mortality of the patients. A group of five cases from the Cleveland Clinic is reported to emphasize the peculiar surgical features presented by these tumors and the manner in which they may best be handled.

The gross appearance of the carotid body was first described by von Haller[1] in 1743, and its microscopic characteristics by Luschka[2] in 1862. A tumor of the carotid body was first reported by Marchand[3] in 1891. Scudder[4] was the first in the United States (1903) to resect this type of tumor and to describe it in American literature.

According to reports in the literature[5-7] and in our own experience, the average age of onset of this type of neoplasm is forty years, extremes ranging from the ages of fifteen to sixty-eight with a slight preponderance in the female.

The normal carotid body is approximately 5 mm. in length, reddish-brown in color and enveloped by a fibrous capsule. It contains chromaffin cells and is, therefore, a member of that loosely classified group, the chromaffin system. It is located immediately posterior to the bifurcation of the common carotid artery.

The chromaffin cells are ectodermal in origin, arising from the neural crest in close association with sympathetic cells.[8,9] The special nerve endings (chemoreceptors) found in the carotid body send afferent impulses via the carotid branch of the ninth (glossopharyngeal) cranial nerve.

The principal pathologic condition of the carotid body is tumor formation. The tumors are usually closely adherent to the carotid vessels and surrounding structures and vary in size from 3 to 15 cm. These tumors are richly vascular as one would suspect from their reddish hue and pulsatile nature. Short vessels stem from the carotid arteries directly into the mass, rendering excessive hemorrhage or rupture of a major vessel likely possibilities. Vertical as well as horizontal extension occurs and in many cases the mass presents in the pharyngeal or tonsillar region.

In the benign type of tumor a definite capsule is usually evident. Malignant changes occur in 15 to 20 per cent of patients but metastases are usually confined to the regional lymph nodes and surrounding tissue. Distant metastases are very rare. One of the few proved cases of such a process is included in the following case reports.

The microscopic appearance is characterized by a loose, syncytial arrangement including numerous blood vessels and cavernous sinuses. The cells are polyhedral with granular cytoplasm and chromaffin granules can usually be demonstrated by special staining technics. Some pericapillary grouping of cells occurs.

Schmidt and others[10,11] have demonstrated rather conclusively that the carotid body functions as a chemoreceptor, being sensitive to carbon dioxide increases, anoxemia and the cyanide radical. Stimulation produces an increase in rate and depth of respiration. The chemoreceptor mechanism does not play an active rôle in respiration under ordinary conditions but is a primitive method of preventing respiratory failure as the respiratory center *per se* is not stimulated by anoxia.

SYMPTOMS

The characteristic picture is that of a slowly growing, painless, non-tender mass

in the superior anterior cervical triangle beneath the border of the sternocleido-mastoid muscle. The mass is laterally mobile about the axis of the carotid arteries but infiltration of local structures prohibits vertical motion.

Depending upon the degree of vascularity, bruit, thrill and expansile pulsations may be demonstrated. Compression and irritation of local structures frequently occurs, producing sympathetic, vagal and glossopharyngeal nerve damage, resulting in Horner's syndrome, vocal cord paralysis and atrophy of the tongue.[12,13] The carotid sinus syndrome occurs in approximately 3 per cent of cases.[14]

In the differential diagnosis one must consider branchial cleft cyst, tuberculous lymphadenitis, aneurysm, cystic hygroma, lymphoma, metastatic carcinoma or sarcoma, aberrant thyroid tissue and abscess.

TREATMENT

The use of irradiation therapy in the treatment of carotid body tumors has been almost universally unsuccessful. A few reports, however, are encouraging.[14,15] Both benign and malignant varieties respond slightly, if at all, and operative difficulties are further increased when surgery is finally undertaken.

The surgical difficulties encountered are mainly those of inaccessibility of the tumor, many extending beneath the mandible to present in the pharynx or tonsillar fossa and hemorrhage.

The most satisfactory approach has been through a wide incision over the long axis of the tumor with careful sharp dissection along the cleavage plane separating the mass from the adherent large vessels. Local coagulants have proved to be of value in these cases.

Ligation of the common carotid artery is not to be undertaken lightly; a mortality of 24 to 65 per cent[15,16] results from such a procedure. Complications occasioned by cerebral ischemia, such as hemiplegia and aphasia, occurred in over 7 per cent, while damage to the jugular vein, recurrent laryngeal nerve, hypoglossal nerve, facial nerve or sympathetic trunks occurred in 24 per cent of 148 operated patients.[16]

In this group it was necessary to ligate the common carotid artery in approximately 50 per cent of the patients, producing a mortality rate of 30 per cent. About 50 per cent of the survivors suffered permanent brain damage.[1]

Periodic compression of the artery for several weeks prior to operation might feasibly stimulate collateral cerebral circulation to an appreciable extent and decrease the complications resulting from ligation.

From our experience and from case reports in the literature[5,6,16,17,18] it has been brought out that 80 or 85 per cent of carotid body neoplasms are benign and slow-growing. By far the large percentage of the malignant variety recur or metastasize only locally.

CASE REPORTS*

Case I in this series is one of the very few, if not the only case, proved to have had distant metastases from a carotid body tumor. The metastasis in the vertebral bodies was visualized during both operations and biopsies taken each time. The tissue proved to be identical in microscopic appearance to that previously removed at the time of the cervical operation. X-ray evidence of distant generalized bone metastases was also present.

CASE I. A white man, aged twenty-seven, was first seen by Dr. George Crile, Sr., in 1906, complaining of a painless, progressively enlarging left cervical mass. This was resected along with a part of the internal jugular vein. (Fig. 1A.) Recovery was uneventful except for a left Horner's syndrome and twelfth cranial nerve paralysis.

The patient was then free of symptoms until January, 1920, when he developed paraplegia and anesthesia to the level of the umbilicus and was admitted to the hospital. Laminectomy

* Case I was partially reviewed and case IV was reported by A. Graham[19] in 1913. They are being represented to reveal more recent developments.
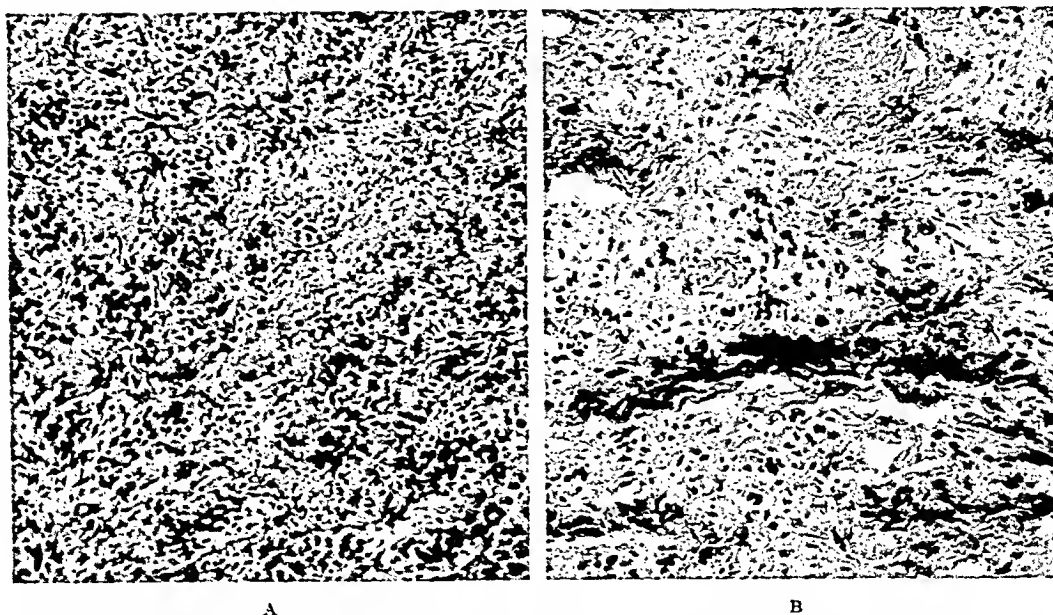
Fig. 1. A, photomicrograph of cervical neoplasm removed in Case 1. Note the loose syncytial arrangement. × 100. B, photomicrograph of biopsy specimen removed at time of second laminectomy of Case 1. × 100.

revealed an extremely vascular tumor jutting from the fifth and sixth thoracic vertebrae on the left and compressing the spinal cord. The local musculature, bone and soft tissue were extremely vascular. Only part of the tumor could be removed because of the extensive infiltration of bone and soft tissue already present. The patient made an uneventful recovery and was not seen again until his readmission in March, 1931.

At that time he complained of pain and progressive instability of the left knee of four years' duration. Imperfect sphincter control and paresthesia of the left sole had been present for one year. Physical examination revealed a pulsating mass, 6 cm. in diameter, in the region of the manubrium sterni. Distinct bruit was present over this area. Marked tenderness in the region of the right iliac crest and muscular atrophy of the left leg were present.

A left Horner's syndrome, twelfth cranial nerve paralysis (results of resection of cervical tumor), spastic gait, hypesthesia below the eighth thoracic segment and a bilateral Babinski sign were present. Distinct bruit was noted over the site of the laminectomy.

Lumbar puncture at L5-S1 revealed a complete block. The fluid was faintly yellow, Pandy 2 plus, protein 130 mg., cells 5, colloidal gold 1112222000 and Wassermann reaction negative.

X-ray examination revealed multiple tumors of the eighth and ninth ribs on the right and of the fifth rib on the left. Rarefaction of the manubrium without definite osteogenesis was present while an osteogenic lesion of the right ilium was noted. X-ray therapy to the bone lesions afforded no symptomatic relief.

On May 20, 1931, the laminectomy site was reopened and a firm, elastic, dark red mass measuring 5 by 1.5 cm. was found protruding from the bodies of the fifth and sixth thoracic vertebrae. The section of tumor compressing the spinal cord was resected with much bleeding. (Fig. 1B.)

The patient recovered sufficiently to be discharged from the hospital but died on July 29, 1931. Autopsy was not permitted.

CASE II. A woman, aged forty-seven, was first seen by Dr. George Crile, Jr., on July 8, 1946, complaining of a small, moderately firm nodule which had appeared in the region of bifurcation of the left common carotid artery in April of the previous year. The mass had never been tender nor had signs of inflammation occurred but a gradual increase in size had been noted.

Physical examination revealed a mass, 4 cm. in diameter, extending beneath the angle of the mandible on the left. The tumor was laterally but not vertically mobile. There was no bruit, tenderness or sign of inflammation. Pressure over the area produced no alteration in blood pressure or respiration. The remainder of the physical examination and laboratory work was essentially negative.

On August 3, 1946, under intravenous pentothal anesthesia a vertical incision was made over the long axis of the tumor. The mass was
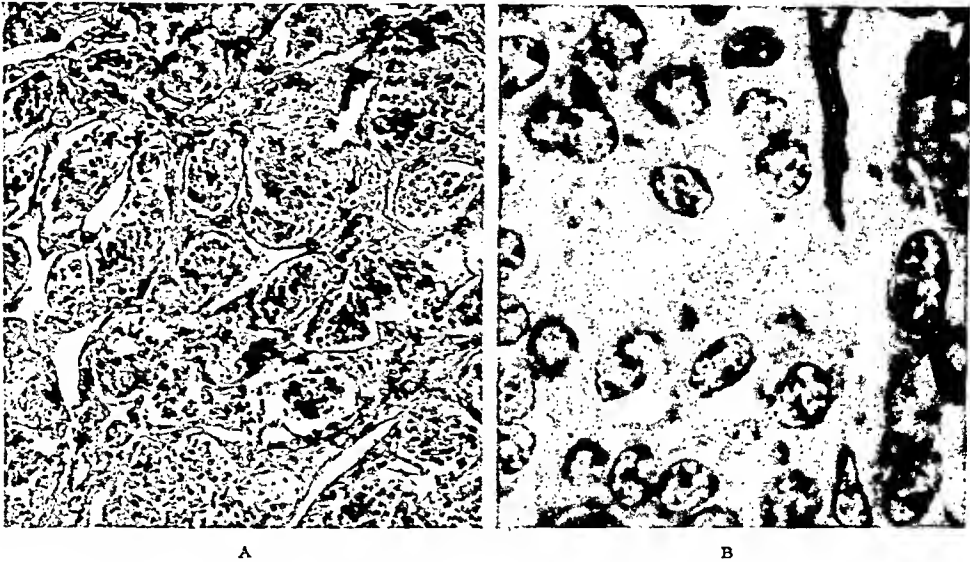
Fig. 2. A, photomicrograph of carotid body tumor removed from Case II. Note the tremendous vascularity and almost sinusoidal character of some of the vessels. Cell grouping and tendency to alveolar formation is rather pronounced. × 100. B, photomicrograph of Figure 2A. Note granular cytoplasm of the cells × 1000.

reddish-brown in color, closely adherent to the carotid arteries in the region of bifurcation and extremely vascular as were the surrounding tissues. No satisfactory avascular cleavage plane could be defined. There was profuse bleeding from the tumor, from the neighboring tissues and from short vessels leading directly from the carotid arteries into the neoplasm. The entire mass was removed and the patient made an uneventful recovery except for transient pain along the distribution of the left posterior auricular nerve.

The gross specimen was globular, rubbery and measured 3 by 2.6 by 1.8 cm. On section the neoplasm was moderately fibrous and firm with a reddish-yellow, irregular central area. Microscopic examination revealed a typical picture of carotid body tumor with large, blood-filled sinuses, loosely arranged stroma and rare mitoses. (Fig. 2.)

CASE III. A white man, aged twenty-seven, was admitted on March 12, 1913, complaining of a small, painless mass which had appeared in the region of bifurcation of the right common carotid artery seven years previously. The mass had gradually increased in size but there had been no pain or signs of inflammation at any time.

Physical examination revealed a well nourished, well developed white man with a mass in the right cervical region extending from beneath the angle of the jaw. The tumor was rounded, smooth, not tender or inflamed and

approximately 6 cm. in diameter. It was mobile laterally but not vertically. The mass completely filled the right tonsillar fossa and encroached on the uvula. Slight enlargement of the right submaxillary glands was present. The remainder of the examination and laboratory work was essentially negative.

The tumor was removed by Dr. George Crile, Sr., and the postoperative course was uneventful.

A thin, loose, fibrous capsule surrounded the reddish-brown, hemorrhagic mass. Microscopic examination demonstrated the characteristic picture of carotid body tumor.

CASE IV. A white woman, aged fifty-six, was admitted to the Clinic on July 8, 1929, complaining of a mass which had been present in the left cervical region for fifteen years. The mass was painless, not tender and had gradually increased in size so that during the previous five years some pharyngeal obstruction had occurred.

In 1920 while "scraping mucus" from her throat, she had initiated a severe hemorrhage. After ten repeated hemorrhages the carotid artery was ligated at another hospital. The patient had five rather severe pharyngeal hemorrhages thereafter.

Physical examination revealed an emaciated, white woman who appeared to be in excess of the stated age. A large mass was present in the left cervical region and presented also in the left tonsillar fossa, producing a high degree of

obstruction. The remainder of the general physical examination and laboratory examination were essentially negative.

On July 15, 1929, the patient was anesthetized with nitrous oxide and oxygen in preparation for resection of the tumor. Due to marked respiratory distress a tracheotomy was performed and removal of the tumor was not attempted.

Four days later Dr. T. E. Jones made a transverse incision over the tumor and divided the sternocleidomastoid muscle in the upper third. The tissues surrounding the tumor contained numerous, large tortuous arteries and veins. Considerable hemorrhage was encountered and several transfusions were necessary. The entire mass, measuring 7 by 6 by 2 cm., was finally removed but it was necessary to leave packing in the wound to control the hemorrhage.

The tumor was reddish-brown in color, had a thin capsule with many dilatated blood vessels on the surface and was moderately soft and rubbery in consistency. The cut surface was grayish-yellow in color with many areas of old and recent hemorrhage. A marked fibrous stroma was present. The cut surface was translucent, irregularly lobulated and traversed by many large blood vessels.

Microscopic examination revealed a very vascular bed with loose cellular tissue having no definite arrangement. Some areas of solid clumps of cells with a tendency toward alveolar arrangement were present. Cells were of variable size with deeply staining nuclei and diffusely granular cytoplasm. There was no evidence of nerve trunks or ganglion cells. Areas of extensive diffuse hemorrhage were present in various parts of the tumor. The patient ran a rather stormy postoperative course but recovered completely.

CASE V. A white man, aged forty-eight, was admitted to the hospital on August 5, 1924, complaining of a soft mass which had been present in the left cervical region for four to five years. The mass had gradually increased in size, especially during the previous six months, and for the past three to four months moderate tenderness had been present.

Physical examination revealed a rubbery tumor, approximately 6 cm. in diameter, in the area of bifurcation of the left common carotid artery. The mass presented in the left posterior pharyngeal fossa. The mucosa was not affected.

The remainder of the physical examination and laboratory findings were essentially negative.

At operation a slightly lobulated mass enveloping the common carotid artery was removed by Dr. George Crile, Sr. The tissue was reddish-brown, somewhat friable and very vascular. On microscopic examination typical features of a carotid body tumor were noted.

Recovery was uneventful except for a minor wound infection.

SUMMARY

1. Removal of tumors of the carotid body is difficult and is associated with high morbidity and mortality.

2. The carotid artery should not be sacrificed unless its ligation is necessitated by uncontrollable hemorrhage.

3. If patience and care are used, most carotid body tumors can be removed without damaging the carotid artery.

4. Irradiation therapy is not often effective in arresting the growth of carotid body tumors.

5. Five cases of carotid body tumor are reported.

6. In one of these cases the tumor metastasized to bones.

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MANY malignant tumors fail to metastasize. For example, two types of tumors both of which have the same microscopic pattern and react in a similar manner to the tissues in which they originate, may act in manners far different; one may metastasize and the other remain localized. No doubt metastasis is influenced by resistance.

From "Metastases Medical and Surgical" by Malford W. Thewlis (Charlotte Medical Press).

EFFECT OF INTESTINAL GASES UPON BALLOONS OF INTESTINAL DECOMPRESSION TUBES*

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THAT rubber membranes were permeable to gases has been known to physicists for over one hundred years. This fact was first noted in 1831 when Mitchell published a paper[1] discussing the permeability of rubber membranes to gases. He noted at that time that the rate of permeability varies with different gases used. Concomitant with the tremendous strides made in the development of synthetic rubbers it was noted that the different rubbers behaved differently to each specific gas. That is, it was soon noted that the permeability of each type of synthetic rubber differed for each kind of gas. Extensive studies were carried on throughout the world with the final result that the permeability of each type of rubber has been charted for each specific gas under many conditions of temperature and pressure.

As a result of studies by numerous workers[2,3,4] it has been demonstrated that the behavior of gases in rubber is similar to that of gases in organic liquids. As a result of this observation rubber may be considered as an organic liquid of high molecular weight. Thus when a gas is brought into contact with a rubber membrane it goes into solution in the rubber on the one side of the membrane and then emerges on the other side by evaporation. It is quite important to note that this process of permeation consists of two separate and distinct factors: first, the solubility of the gas into the rubber and second, the diffusion of the gas through the rubber and its evaporation on the other side of the membrane. In studying this phenomenon it was soon noted that the permeability of any specific gas through a rubber membrane is independent of the presence of any alien gas on the other side of the membrane. In addition, the law of diffusion of gases applies to the passage of the gas through the rubber membrane, namely, any specific gas tends to pass from an area of increased pressure into one of decreased pressure for that specific gas until equal pressures for that gas are obtained on both sides of the rubber membrane. In addition, the presence of other gases on the one side of the rubber membrane has no effect upon the permeability of the rubber to any one specific gas.

The results of these extensive researches were finally formulated into two laws: Henry's law which postulates that the gas first dissolves in the rubber to a degree which is in proportion to the pressure, and Frick's Law which postulates that the gas dissolved in the rubber diffuses in the rubber toward that part of the rubber where the gas is present in a lower concentration; finally, the gas evaporates out of the rubber.

Of all the gases found in the gastrointestinal tract in cases of intestinal distention, carbon dioxide and hydrogen sulfide are apt to be most troublesome for the intubator. The reason for this is that these gases are highly permeable to the natural rubber found in the balloons of the long intestinal decompression tubes in use today. In addition, the concentration of the gases within the bowel is greater than their concentration within the balloon of the intestinal decompression tube. It should be quite evident that although we express all the atmospheric air from the balloon of the intestinal decompression tube before insert-

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ing it, nevertheless some atmospheric air remains within the balloon. From the preceding discussion, following the law of diffusion of gases one would expect the carbon dioxide to diffuse through the wall of the balloon into its lumen distending it until the pressure of the carbon dioxide within the balloon equalled its pressure around it, i.e., in the intestinal tract. The same mechanism would of course apply to the other gases found in the gastrointestinal tract. However, as noted previously, the other gases, except hydrogen sulfide, are not nearly as readily diffusible through a rubber membrane. For this reason, the authors began their investigative study upon the balloons of intestinal decompression tubes by using carbon dioxide.

The importance of these studies is quite apparent when one realizes that an increase in the amount of gas within the balloon of the long intestinal decompression tube, where no gas is supposed to be, results in a bulging of the bowel wall around it. If the patient is partially obstructed, a ball-valve type of obstruction results from the presence of a distended balloon pushing against the point of obstruction. If the patient is suffering from stomal edema at the point of anastomosis, the presence of such a distended balloon would completely obstruct the bowel. On the other hand, when an attempt is made at the removal of a tube with a distended balloon, a tear in the anastomosis may occur if the tube head is distal to the anastomotic site. In some cases, the balloon may become so tremendously distended as to tear the bowel. A case in which the metal tip of a Miller-Abbott tube has perforated the bowel at the point of obstruction has been reported.[5] These accidents, although uncommon, have been noted with long intestinal decompression tubes. A knot formation in the Miller-Abbott tube proximal to the balloon may set the stage for the development of a balloon markedly distended with gas.[6]

As a result of our studies upon the effect of intestinal gases upon the balloons of

intestinal decompression tubes, we have succeeded in completely eliminating this complication to intestinal intubation. The construction of the Cantor tube lent itself very well to preventing this complication simply and easily; whereas the use of the Miller-Abbott tube, which depends upon an air-filled balloon alone, cannot be so treated to prevent the introduction of gas into the balloon if the channel for inflation becomes obstructed. In the use of any other tube depending upon mercury for its downward passage, prevention of the intake of gas by the balloon is possible as we shall demonstrate in our experimental studies.

EXPERIMENTAL STUDIES

The gas used in our studies was carbon dioxide because, as noted, it is the most diffusible through rubber membranes and because its concentration within the bowel increases with the duration of the bowel obstruction. We tested this gas with all the commonly used types of intestinal decompression tubes. The apparatus used to test the effect of the gas upon the balloons of intestinal decompression tubes was a very simple yet effective one. Figure 1 demonstrates the gas cylinder with gauge capable of permitting the gas to enter our pressure chamber "p" at a definite rate to compensate for the loss of gas through the rubber connections and the incomplete gas-tightness of our equipment. The pressure chamber "p" is then connected to a mercury manometer "m" which measures the pressure within the chamber correctly to the millimeter of mercury. After much experimentation we were able to set our flow of gas into the chamber at such a rate as to keep the pressure of the gas in the pressure chamber constant within fairly narrow limits. In this way we were able to subject our tube heads to varying degrees of pressure at room temperature. In order to insure that the chamber was filled with pure gas being tested, at the beginning of each experiment we would place the tube heads within the chamber, place the lid

upon the pressure chamber, then using the blade of a thin knife under the lid we would turn on a full flow of the pure gas into the chamber at high pressure. If this is done for several minutes, the air present in the chamber is forced out through the chink made by the blade of the penknife. At the end of several minutes the gas pressure going into the pressure chamber is reduced and the knife blade is quickly removed. Our pressure chamber now contains a pure gas from the cylinder. We now set the flow of gas into the chamber at such a rate as to keep the pressure within the chamber at any desired level.

The volume of the gas within the balloons was determined by the water displacement method. By immersing our balloons into a measured amount of water in a measuring flask of 250 cc. capacity, we were able to determine rapidly and simply the amount of gas within the balloon. The value obtained for each balloon before being put into the pressure chamber (with balloon empty) when subtracted from the value obtained after removal of the balloon from the pressure chamber would be the amount of gas taken up by the balloon in cubic centimeters.

In using all three types of intestinal decompression tubes commonly used today, we noted first that the balloons were all of latex rubber so that the diffusibility of any specific gas through this specific rubber should be the same under the same conditions. However, on measuring the thickness of the walls of the balloons used with the Miller-Abbott and Harris tubes, we found them to be 0.006 mm. thick. The thickness of the wall of the Cantor tube ranged from 0.024 mm. in its middle to 0.040 mm. at the base and neck. In addition, the Cantor tube and the Miller-Abbott tube both present balloons about $2\frac{1}{2}$ inches long with the diameter of the Miller-Abbott tube somewhat greater than the balloon of the Cantor tube. The balloon of the Harris tube, however, is 6 inches long and of the same diameter as the Miller-Abbott. Referring to the formula quoted by van
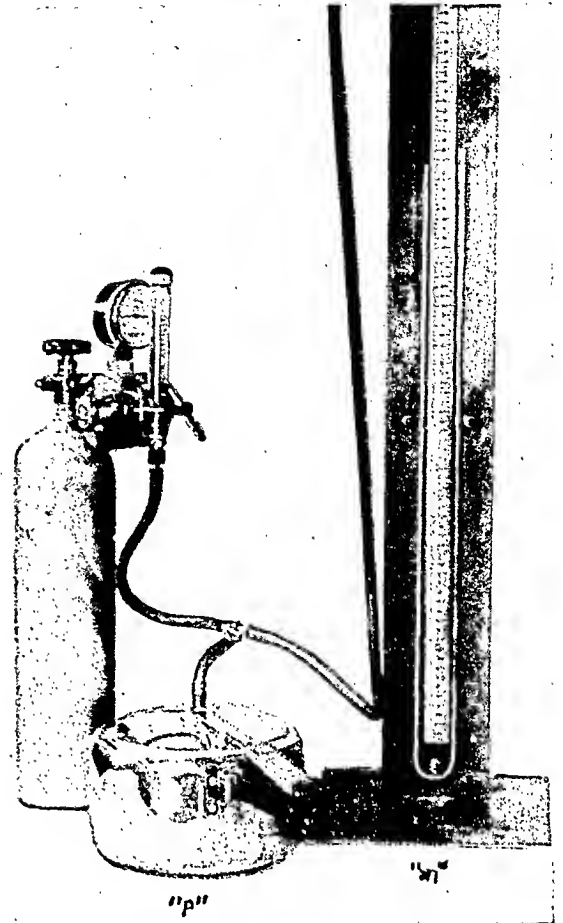
Fig. 1. Apparatus used in conducting the experiments; "p" is gas chamber in which balloons were placed and subjected to a constant pressure of carbon dioxide gas; "m" manometer measuring the gas pressure within the pressure chamber.

Amerongen[7] for the quantity of gas permeating through a rubber membrane we find:

$$q = DbA(p_1 - p_2/d)t$$

Since "d" represents the thickness of the rubber membrane, it would be evident that the balloons of the Miller-Abbott and Harris tubes would be expected to take up more gas than the balloon of the Cantor tube which is almost four times as thick. Moreover, the Harris tube having a balloon 6 inches long would be expected to take up far more gas than any other tube in use today, because the value for "A" which is the area of the rubber membrane is more than double that of either the Miller-Abbott or Cantor tube. In the equation quoted above, q is the quantity of gas permeating through the specific rubber. "D" is the diffusibility of each specific gas

FIG. 2. The balloon containing the mercury also contains a well defined amount of gas. Note position of balloon at the duodenojejunal flexure.

FIG. 3. Note that now the balloon is at the ileocecal valve; note also that the amount of gas within the balloon has diminished.

through each specific type of rubber membrane. This value has been determined for all types of rubber and all types of gas. "h" is the solubility of the gas in the specific type of rubber membrane. This value has been determined for all types of rubber and all types of gases which might be found in the gastrointestinal tract. "$p1 - p2$" is the pressure of the specific gas on each side of the rubber membrane; "t" is the time for which the rubber membrane is exposed to the gas.

With these preliminary observations in mind, we proceeded to test all the tube heads under varying conditions to check the applicability of the formula quoted by van Amerongen to clinical medicine.

Experiment 1. To note the effects of high pressures upon the balloons of intestinal tubes, we placed the sealed empty balloons into the chamber and raised the pressure of the carbon dioxide to 180 mm. of mercury for twenty-four hours. At the end of this period of time, we found 10 cc. of gas in the balloons. This rapid observa-

tion informed us that gas does enter the balloons. We then proceeded systematically to study these balloons under varying conditions.

Experiment 2. For this experiment three balloons from Cantor tubes were used. The balloons were used without being attached to the rubber tubes in order to determine the effect of the carbon dioxide upon the balloons by themselves without tubal attachment. Two of the balloons were tied off at the neck using a silk tie and one balloon was cemented closed at its mouth. In this fashion we could determine whether the presence of the silk tie in any way influenced the permeability of the balloons. It was thought that the tie might injure the rubber so as to increase its permeability at that point. In this experiment a pressure of 37 mm. of mercury was maintained in an atmosphere of carbon dioxide for twenty-four hours. At the end of that period of time it was found that the cemented balloon contained 7 cc. of carbon dioxide whereas the tied off balloons con-
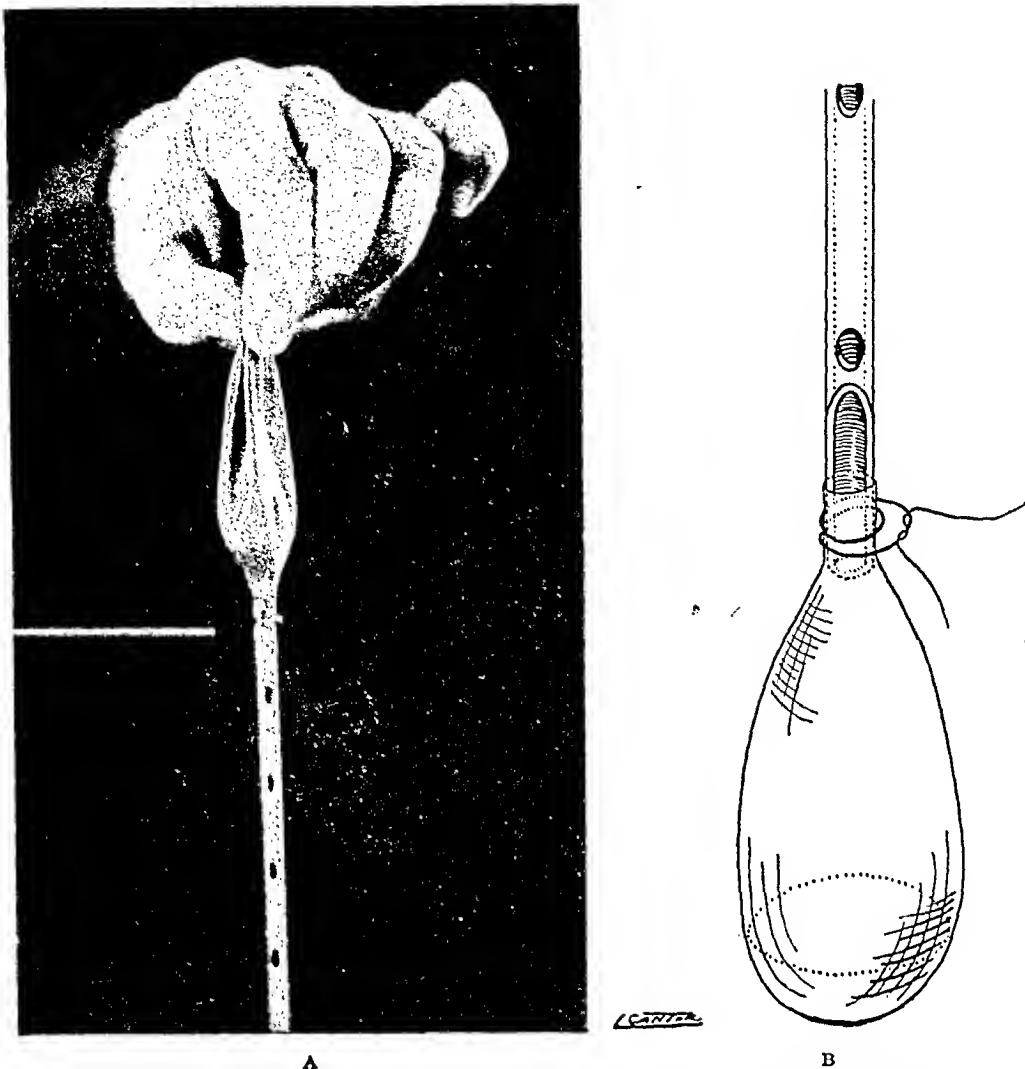
FIG. 4. A, the tie is applied to the shaft of the tube over the point of attachment of the balloon. This tie is tied tight enough to trap the mercury but air can get in and out. To accomplish this, insert a stylet from a No. 22 gauge needle through the last hole into the balloon and tie over it. After tying off the tube remove the stylet. This will leave a small opening through which gas can come out but no mercury. B, sketch of balloon attached to the tube. Note double loop tie being applied to the point of attachment of balloon to tube.

tained 9 cc. for one and 3 cc. for the other. We examined the tied off balloons carefully to determine the reason for the variation in the amount of gas. We concluded from our examination that the tightness of the tie was the important factor. In that balloon in which the tie had been looser the highly diffusible carbon dioxide readily escaped whereas in the very tightly tied balloon it remained trapped. This at once suggested a preventative to the development of gas within the balloon of the Cantor tube when clinically used.

The balloons containing the carbon dioxide were then permitted to lie exposed to the atmosphere to determine how rapidly the

carbon dioxide would diffuse out, since the concentration of carbon dioxide in the atmosphere is much lower than that now present in the balloon. At the end of forty-five minutes we found that the cemented balloon had lost 2 cc. of the gas, the tightly tied balloon had lost 3 cc. of its gas whereas the balloon not tied so tightly had lost all of its gas.

The same balloons were permitted to lie exposed to the atmosphere overnight and were rechecked in the morning. At this time no gas was found in any of the balloons.

The practical implications of these observations are apparent. If for any reason

the balloon of the long intestinal decompression tube should become torn off and gas remain trapped within the balloon, by decompressing the bowel the gas would rapidly leave the balloon so that the balloon containing mercury could move down the gastrointestinal tract and so be excreted. Figures 2 and 3 present an unusual case in which this accident occurred. After being in the patient twelve days the balloon was torn from the tube during the removal of the intubation tube. It can readily be seen from Figure 2 that a fairly definite amount of gas is present in the balloon which can be identified as being at or just beyond the duodenojejunal flexure. Figure 3 was taken four days after Figure 2. The amount of gas present in the balloon has decreased, and the balloon containing the mercury has moved down to the ileocecal valve. This experiment was repeated three times with the same results being obtained.

To prevent any possible chance of this happening again, we advise that the tie in the Cantor tube be placed on the shaft of the tube just above its distal end at the point at which the balloon is cemented to the tube. (Fig. 4.) By so doing, it would be almost impossible to tear off the balloon; and if it did occur, the gas and mercury would readily escape without harm to the patient.

Experiment 3. In this experiment ten balloons were used to determine the effect of a pressure of 15 mm. of mercury in an atmosphere of carbon dioxide for four days. Both new and used balloons were utilized in this experiment. This study would demonstrate any changes in permeability that the balloons might undergo by virtue of having been soaked in the intestinal contents of the patient for varying lengths of time as well as the effect of the mercury upon the rubber of the balloons:

Balloon 1 was a new balloon which was tied off with 5 cc. of mercury for five weeks. At the end of this period of time the rubber of the balloon was discolored black. There was no vaporization of the mercury in the balloon since the volume of this balloon and the volume of a new balloon containing the same amount of mercury was exactly the same. At the end of the four days at 15 mm. of mercury carbon dioxide gas pressure, this balloon took up 7 cc. of carbon dioxide.

Balloon 2 had been in the patient soaking in intestinal secretions for twenty-four hours. This balloon was then dried and sealed empty in an envelope for thirteen more days before being used in this experiment. Care was taken to tie off the neck of the balloon tightly and to separate the walls of the balloon so they would not stick. This balloon took up 23 cc. of carbon dioxide in the four-day period at 15 mm. of mercury.

Balloon 3 had been in the patient for five days soaking in intestinal secretions. The balloon was then dried and kept in a sealed envelope for thirteen more days before being used in this experiment. This balloon took up only 4 cc. in four days at 15 mm. mercury. It is apparent from this that soaking the balloon in the intestinal secretion does not increase its permeability but that some other explanation must be sought for the result in balloon 2. We believed that the probable answer was that in balloon 3 the walls of the balloon were squeezed together in an effort to express all the atmospheric air with the result that they tended to stick together so that the two sides of the balloon acted as one membrane with no cavity into which the carbon dioxide could diffuse. This point will be checked again in experiment 4.

Balloon 4 was in the patient for ten days soaking in the intestinal secretion, and the mercury remained in the balloon for a total of thirty-seven days. This balloon took up 15 cc. of carbon dioxide in the four days at 15 mm. of mercury pressure.

Balloon 5 was in the patient for five days and in a sealed envelope for thirteen days more. No mercury was used in this balloon during the test. This balloon took up 4 cc. of carbon dioxide.

Balloon 6 from a Miller-Abbott tube was tied off at both ends without a tube passing through it in an effort to see whether the empty balloon itself would take up any carbon dioxide. We found no carbon dioxide in this balloon at the end of four days at 15 mm. mercury pressure. The walls of the balloon were in close contact with each other because of their thinness. The question arose as to whether this balloon was so highly permeable that it could not retain any carbon dioxide or whether, as in balloon 3, the walls of the balloon being in contact with each other there was no space into which the gas could pass. We, therefore, investigated this point in experiment 4.

Ballon 7, an empty latex balloon from a Harris tube, was then tied off at each end. This was a new balloon. This balloon did not take up any carbon dioxide. Again the question arose as to whether the balloon was so highly permeable that it could not hold the carbon dioxide or whether the walls of the balloon in contact with each other prevented the gas from entering. This, too, is to be studied in experiment 4.

Balloon 8 was attached to a 2-inch length of tube. Five cc. of mercury was placed in the balloon and the balloon sealed by tying tightly at the point at which the balloon was cemented on to the tube (Cantor). In this way, the tube was tied off so tightly that neither air nor mercury could get in or out. This balloon took up 18 cc. of carbon dioxide in the four-day period at a pressure of 15 mm. of mercury. From this it is evident that the mercury by separating the walls of the balloon creates a space into which the carbon dioxide could diffuse.

Balloon 9 was attached to a 2-inch length of tube and 5 cc. of mercury placed in the balloon. The tube was tied off at the same point as in balloon 8, but the tie was tied only tight enough to trap the mercury but not tightly enough to seal in the air. We could blow air into the balloon and squeeze it out of the balloon but the mercury could not escape. By squeezing all the air out of

this tied off balloon and placing it into the same chamber for the same period of time as balloon 8, we found that this balloon took up only 6 cc. of carbon dioxide. Furthermore, when this balloon was immersed in our measuring flask of water to determine the volume of carbon dioxide which it took up, the gas rapidly bubbled out of the balloon due to the slight pressure of the column of water around it.

Balloon 10 was attached to a 2-inch length of tube. Five cc. of mercury was placed in the balloon. The tube was then tied off at the same point as balloons 8 and 9 (at the distal end of the tube just above its terminal end and at the point at which the balloon was cemented on). The double loop tie was again tied tightly enough to seal in the mercury but the air could readily get in and out of the balloon. This balloon was found to take up 8 cc. of carbon dioxide at the end of four days at a pressure of 15 mm. of mercury. This balloon also rapidly lost its carbon dioxide by the slight water pressure incident to measuring its gas volume.

Conclusions from This Experiment. It is evident from this experiment that the length of time that the balloon is permitted to soak in gastrointestinal contents does not affect its permeability, nor does the length of time that the mercury is permitted to remain in contact with the rubber appear to effect its permeability to carbon dioxide. It also suggests that the walls of the balloon must be separated before the balloon will take up any carbon dioxide although this point will be checked in experiment 4. The most important observation in this experiment is that when the tie is applied tightly enough so as to trap the mercury in the balloon, yet not so tight that air can get in and out of the balloon, a markedly reduced volume of carbon dioxide is taken up and this small volume is rapidly lost with the slightest pressure. Clinically, a practical application of this point would be the application of the tie in the Cantor tube to the point just above the terminal end of the tube at which the balloon is
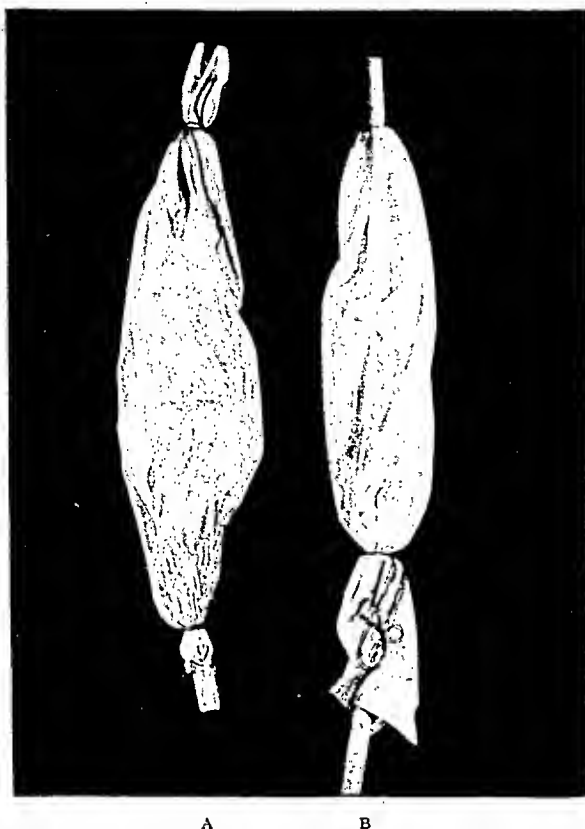
FIG. 5. Balloons of Harris tubes. A, balloon containing mercury; B, no mercury in this balloon. Note that both balloons contain gas.
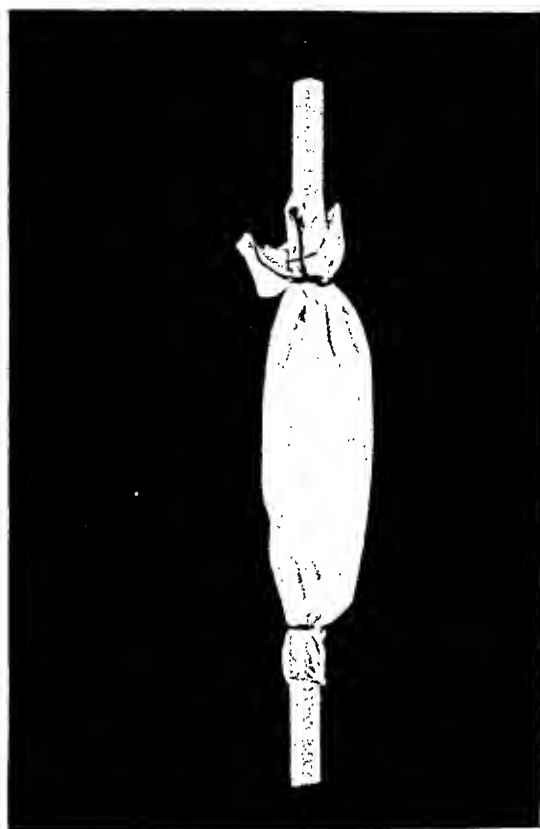
FIG. 6. Balloon of Miller-Abbott tube; note gas in balloon.

cemented on. (Fig. 4.) If this tie is tied tightly enough to trap the mercury but not completely occlude the tube so that air can get in and out, not only will such a tube take up a very small amount of gas if exposed to such high pressure, but the contraction of the bowel wall upon the balloon and the pressure of intestinal secretions would promptly empty the balloon of all gas. In this fashion by applying one tie in this way, it would be impossible for any gas to remain trapped in the balloon of the Cantor tube. This same point might be utilized in other types of tubes that depend upon mercury alone to carry the tube head downward. It would not be used in the Miller-Abbott tube in which air alone is used to fill the balloon or in any air and mercury tube as such tube heads must be airtight. In the Miller-Abbott tube, however, the double lumen which permits aspiration of the distended balloon would furnish an adequate safety valve. In the

event that the tube became knotted, however, the gas would be trapped and the balloon would then distend markedly.

Experiment 4. In order to determine whether the latex rubber of the Miller-Abbott and Harris tubes with a thickness of 0.006 mm. was so highly permeable to carbon dioxide that it could not retain it or whether the walls of the balloon in experiment 3 were adherent and thus prevented the intake of carbon dioxide, the following experiment was performed:

The balloons were fastened upon rubber tubes exactly as the tube appears for use clinically. All the air was expressed from the balloons and then the tubes with the balloons attached were subjected to a pressure of 34 mm. of mercury in an atmosphere of carbon dioxide for forty-eight hours. At the end of this period of time, the following results were obtained: (1) The Harris tube with mercury in the balloon took up 20 cc. of carbon dioxide. (Fig. 5.) (2) The Harris tube with the
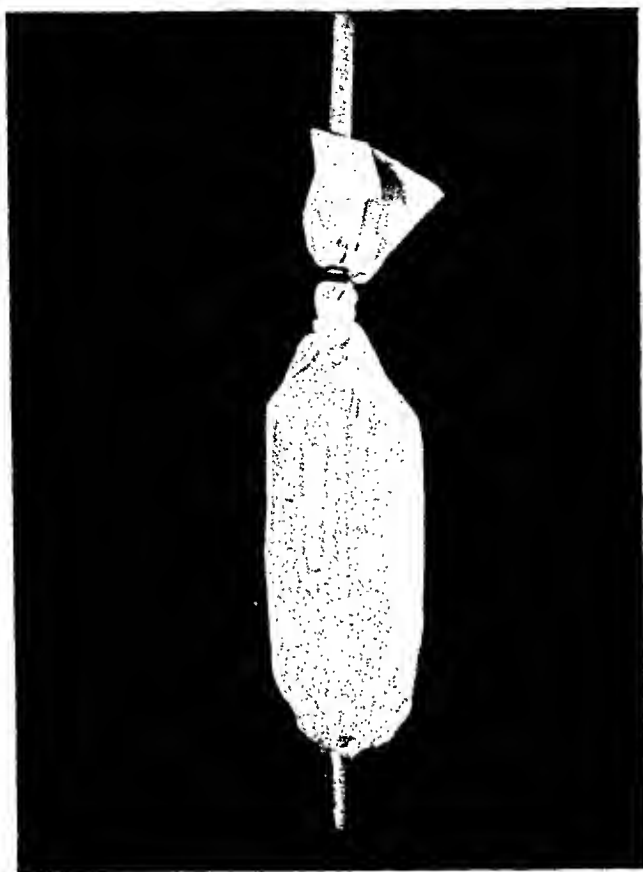
Fig. 7. Balloon of Harris tube containing glass beads. Note large amount of gas in the balloon. This gas was then removed and tube re-tied. This perfused balloon, when subjected to 15 mm. pressure of carbon dioxide, then took up an equal amount of carbon dioxide.

Fig. 8. Balloons of Cantor tube. A, empty balloon; B, balloon containing mercury; this took up little gas. C, balloon containing glass beads. This balloon took up 17 cc. of carbon dioxide.

balloon empty took up 12 cc. of carbon dioxide. (3) The Miller-Abbott tube empty took up 8 cc. of carbon dioxide. (Fig. 6.) (4) The Harris tube with beads took up 66 cc. of Carbon dioxide. (Fig. 7.)

This experiment shows rather conclusively that the carbon dioxide gas does diffuse through the balloons of the Miller-Abbott and Harris tubes. It also demonstrates that this occurs when the walls of the balloons are kept apart by the shaft of the tube which traverses the balloons. In addition, the presence of an inert substance such as mercury which further separates the walls of the balloons permits a greater amount of carbon dioxide to enter the balloons.

We now subjected the balloons to a pressure of 100 mm. of mercury of carbon dioxide to determine whether any more gas would enter the balloons at that high pressure in a short period of time (twenty

minutes). We found that the carbon dioxide did not enter the balloons in the twenty-minute period even at that high pressure. We must conclude from this that the carbon dioxide diffuses into the balloons at a rate such that no increase in the carbon dioxide would be noted at this pressure in the twenty-minute period.

Experiment 5. The purpose of this experiment was to determine whether the mercury caused more carbon dioxide to enter the balloon because of some special effect within the balloon or whether it was merely the inert mass of mercury separating the walls of the balloon which resulted in the increased amount of gas intake as compared with the empty balloons.

In this experiment, we used eight balloons and subjected them to a pressure of carbon dioxide that ranged from 30 to 40 mm. of mercury for forty-eight hours. The following results were obtained:

Balloon 1 was a plain, empty Cantor tube and balloon with all the air expressed from the balloon by rolling up the balloon tightly. This resulted in a close coaptation of the walls of the balloon. At the end of forty-eight hours this balloon had taken up no carbon dioxide.

Balloon 2, a plain empty Cantor tube and balloon, was treated the same as bal-

loon 1. No gas was found in the balloon at the end of forty-eight hours. (Fig. 8.)

Balloon 3: Glass beads in an amount equal to the volume of mercury used was placed in the balloon of the Cantor tube and subjected to the pressures of carbon dioxide as noted. At the end of forty-eight hours 18 cc. of carbon dioxide was found in the balloon. (Fig. 8.)

Balloon 4: Glass beads in an amount equal to the volume of mercury was placed in the balloon of the Cantor tube as in balloon 3. At the end of forty-eight hours 17 cc. of carbon dioxide was found in the balloon.

Balloon 5: Mercury was placed in the balloon of the Cantor tube in an amount equal to the beads and the same conditions used. At the end of forty-eight hours 2 cc. of carbon dioxide was found in the balloon. (Fig. 8.)

Balloon 6: A Cantor tube and mercury in the balloon was utilized as in balloon 5. At the end of forty-eight hours, 2 cc. of carbon dioxide was found in the balloon.

Balloon 7: A Harris tube was used without mercury and subjected to the same conditions. At the end of forty-eight hours this balloon was found to contain 66 cc. of carbon dioxide.

Balloon 7a: A Harris tube with the balloon containing 5 cc. of mercury was subjected to the above conditions. At the end of forty-eight hours this balloon was found to contain 32 cc. of carbon dioxide.

Balloon 8: A Miller-Abbott tube with empty balloon was subjected to the same conditions. At the end of forty-eight hours the balloon was found to contain 10 cc. of carbon dioxide.

The observations in this experiment followed the formula of van Amerongen that the amount of gas that permeated through a rubber membrane is directly influenced by the area of the balloon. Hence, the balloons of the Harris tube took up far more gas than did the balloons of the Miller-Abbott or Cantor tubes. Since the latter two balloons are one-half the size of the balloon of the Harris tube, this result

was to be expected. The experiment also showed that mercury as such did not increase the permeability of the balloons. It acted merely as an inert mass keeping the walls of the balloons apart, thus creating a cavity into which carbon dioxide could permeate. The markedly greater amount of carbon dioxide that diffused into the balloons containing the glass beads as compared with the balloons containing the mercury is quite understandable when one considers that by the use of glass beads the walls of the balloon are separated much better and the small spaces between the surfaces of the beads do not permit the walls of the balloons to rest smoothly upon their surfaces. It becomes evident that anything that will effectively separate the walls of the balloons will result in an increased intake of carbon dioxide. Even with the Miller-Abbott and Harris balloons empty the presence of the shaft of the tube passing through these balloons effectively creates a cavity for the intake and storage of carbon dioxide.

Experiment 6. The purpose of this experiment was to determine whether perfusion thoroughly of the balloons with carbon dioxide until their walls were saturated with this gas would prevent the intake of carbon dioxide.

For this experiment eight balloons were employed. These balloons were subjected to 10 mm. of mercury pressure of carbon dioxide for forty-eight hours. This pressure range is well within that found clinically in cases of intestinal distention in humans. The following results were obtained:

Balloon 1: A Harris tube with the balloon containing mercury was found to have taken up 50 cc. of carbon dioxide at the end of forty-eight hours.

Balloon 2: A Miller-Abbott tube was found to have taken up 17 cc. of carbon dioxide at the end of forty-eight hours.

Balloon 3: A Cantor tube containing glass beads in an amount equal to the amount of mercury used was found to

contain 18 cc. of carbon dioxide as the end of forty-eight hours.

Balloon 4: A Cantor tube was utilized with the balloon containing glass beads in an amount equal to the volume of mercury used. This balloon was found to contain 17 cc. of carbon dioxide.

Balloon 5: A Cantor tube was used with the balloon containing 5 cc. of mercury. This balloon took up 6 cc. of carbon dioxide at the end of forty-eight hours.

Balloon 6: A Cantor tube with the balloon containing 5 cc. of mercury was used. This balloon took up 3 cc. of carbon dioxide at the end of forty-eight hours.

Balloon 7: A Cantor tube was used with the balloon empty. This balloon had been perfused and then rolled up to express all the gas preliminary to being tied off. This balloon took up 3 cc. of carbon dioxide in forty-eight hours.

Balloon 8: A Cantor tube was used with the balloon empty. This balloon had been perfused with carbon dioxide and then the gas evacuated by rolling up the balloon and tying off at its neck. This balloon was found to contain 4 cc. of carbon dioxide at the end of forty-eight hours.

The results of this experiment show rather conclusively that perfusing the balloons thoroughly with carbon dioxide so that the wall of the balloon was thoroughly saturated with this gas, did not prevent the permeation of the gas through the wall of the balloon into its lumen. It has demonstrated that the only important antecedent required for the passage of gas into the balloons of intestinal tubes is the presence of any inert mass that would separate the walls of the balloon thus creating a cavity for gas to accumulate. This experiment was repeated three times with the same result.

CLINICAL APPLICATIONS OF EXPERIMENTAL DATA

As a result of our experimental studies we can state definitely that all balloons of long intestinal decompression tubes are permeable to intestinal gases in the fashion as noted by van Amerongen. The permeability for each specific type of rubber membrane and each specific kind of gas has been determined with such exactitude that a formula has been set up. Being reduced to a formula whose factors are known, the quantity of gas that diffused through a specific type of rubber balloon can readily be computed. Carbon dioxide and hydrogen sulfide are the gases most likely to diffuse into the balloons of intestinal decompression tubes because of their high degree of diffusibility and because of the markedly higher concentration of these gases within the bowel as compared with their concentration within the balloon. Nitrogen and oxygen within the bowel do not vary widely from the concentration found within the balloon which contains atmospheric air. As a result the permeation of these latter gases into the balloons of intestinal tubes would not be expected to be very rapid, if at all, because the difference in pressures of these gases within the balloons and in the bowel is not too divergent. Hydrogen sulfide which accumulates in the gastrointestinal tract in a much higher concentration than is found within the balloon of intestinal decompression tubes is a gas which diffuses through the rubber membranes with such speed and so rapidly that it does constitute a problem.

We have noted rather conclusively that perfusion of the balloons with carbon dioxide will not in any way prevent the diffusion of the carbon dioxide through the wall of the balloon and into its lumen according to the law of diffusion of gases.

An absolute prevention to the accumulation of the gas which permeates through the wall of the balloons of intestinal tubes consists merely in applying the tie to the balloon in such a fashion that the mercury remains trapped within the balloon but air can enter and leave. In this case, the slight pressure of the wall of the bowel during peristaltic activity and the pressure of the liquid contents of the gastrointestinal tract is sufficient to evacuate all gas com-

pletely from the balloons. In addition we urge that all balloons be made of neoprene-G. This rubber, neoprene-G, is only 19 per cent as permeable to carbon dioxide as is latex rubber. By these two changes, we can completely prevent any accumulation of intestinal gases within the balloon.

The law of diffusion of gases acting in a medium of a decompressed bowel is an adequate explanation for the rarity of finding gas in the balloons of intestinal decompression tubes clinically. In our experience with the Cantor tube less than 0.2 per cent of the cases intubated demonstrated gas within the balloons. These cases are invariably those in which the tube is kept in a non-decompressed paralytic bowel for longer than five days. This small incidence of occurrence can be completely eliminated in all types of mercury carrying intestinal decompression tubes.

CONCLUSION

1. All balloons of intestinal decompression tubes are permeable to gases, particularly carbon dioxide and hydrogen sulfide, of the bowel when subjected to prolonged intubation.

2. Since the permeability of the balloons is proportional to the area of the balloon, the Harris tube balloons took up far more gas than any other type of balloon.

3. Prolonged soaking of the balloon in the intestinal secretions does not increase its permeability.

4. The presence of the mercury within the balloon for long periods of time does not increase its permeability.

5. The mercury behaves as an inert body within the balloons and keeps the walls apart so that gas can diffuse into the balloons.

6. An absolute preventative to the accumulation of gas within the balloon of mercury carrying intestinal decompression tubes consists merely in tying on the balloon in such a way that the mercury remains trapped but air can get in and out of the balloon.

7. By using Neoprene-G and tying on the balloon of the Cantor tube in this way, gases cannot accumulate within the balloon of the Cantor tube.*

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* The observations relative to the diffusion of hydrogen sulfide are those of Mitchell.[1] The authors are at present engaged in repeating Mitchell's observations especially noting the effect of the hydrogen sulphide upon the balloons of intestinal tubes.

SURVEY OF SOME ASPECTS OF APPENDICITIS

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IF we could return from paradise a hundred years from now and attend a medical society meeting we might well hear a paper on acute appendicitis. Contrary to our thoughts, which at first might be that such a subject has been discussed many times and is one in which we are thoroughly schooled and on which there could be little change, the fact exists that acute appendicitis constitutes more than 50 per cent of the acute abdominal cases admitted to the hospital[1] and that it still carries an unwarranted mortality. Appendicitis is a subject which to the sophomore medical student is a clear-cut disease offering few obstacles in diagnosis and one which to the veteran physician presents difficult problems in differentiation and calls for profound and discriminating judgment. The author does not propose here to review the cardinal symptoms and signs of acute appendicitis nor to review the differential diagnosis.

If the mortality of appendicitis is to be reduced almost to the vanishing point, it is essential that we, as physicians, appreciate the earliest signs and symptoms of the condition. There are two reasons why patients are operated upon late; either the patient may think that the symptoms are not serious enough for medical advice or the medical advisor may think that the symptoms are not typical of appendicitis or not serious enough to demand operation. It is clear that we have no remedy against the first cause, save the education of the public. As long as a stomach-ache calls for a drug store sale of a physic there are going to be a number of late patients with appendicitis and peritonitis admitted to the hospital. In regard to ourselves it is important that we understand that the so-called typical symptoms of appendicitis as given in the textbooks often indicate a somewhat advanced stage of the condition and that it is impossible to say at the beginning of the attack whether it is likely to be mild or severe in type.

It is desirable to diagnose appendicitis before peritonitis has set in or at least before there is any more than that slight amount of congestion of the peritoneum which is commonly associated with any inflammatory process within the gut. The sensory mechanism of the viscera does not allow the patient to localize the pain as that of appendicitis; it is only when inflammation reaches the peritoneum that localization is accurately established.

The different grades of inflammation of the appendix have for many years been well described and understood, although in some quarters there is still a lack of appreciation of the advanced pathological condition often coexistent with initial symptoms. Catarrh of the mucous membrane, parenchymatous inflammation of the whole wall, gangrene of the interior lining or of all but the peritoneal coat, any of these may coexist with symptoms so slight that they may be overlooked by the patient and thought of slight significance by the surgeon. Even rupture of the appendix due to local gangrene may not cause the patient much trouble so long as local adhesions prevent the extension of the mischief. When the appendix ruptures into the general abdominal cavity, in the absence of any protective adhesions or when after being localized the inflammatory process extends, not even the insensitive patient can refrain from seeking advice.

Obstruction of the lumen of the appendix either by concretion, kink, stricture or adhesion is usually accompanied by more acute symptoms. When the appendix

has ruptured the condition is not merely appendicitis but peritonitis, local, diffuse or general as the case may be and the diagnosis to that extent is more complicated. It is here that the case history is frequently of the greatest help in making certain the true cause of such peritonitis.

TABLE I

SURVEY* OF ONE SURGICAL SERVICE 1942 TO 1946. ST. EDWARD'S HOSPITAL

Cases	All Types	Acute Sub-acute Perforated Abscess	Acute Perforated Abscess	Perforated Abscess	Percentage of Acute Cases Ruptured	Deaths
Entire period of mortality.	616 1.78%	564 1.95%	396 2.02%	118 9.32%	18.6%	11
Before penicillin mortality.	301 2.3%	270 2.6%	183 3.8%	40 17.5%	14.8%	7
After penicillin mortality.	315 1.2%	294 1.3%	213 1.8%	78 5.1%	26.4%	4
Percentage decrease in mortality after penicillin.	47.9%	50%	52.-%	70.9%		

* Two thousand three major surgical procedures; 616 (30.1 per cent) appendectomies exclusive of incidental operations performed in conjunction with other procedures. They were classified as follows: acute 278, perforated 105, abscessed 13, subacute 168, catarrhal 4, mechanical 4 and recurrent 44.

Before leaving general considerations the author would like to say a few words regarding the pelvic appendix. The perforated pelvic appendix is one of the most easily overlooked and therefore, one of the most dangerous conditions which may occur in the abdomen. The early symptoms of an attack of pelvic appendicitis are similar to those which occur when the appendix is in the normal position. While the appendix is unruptured and tense, the pain due to distention and peristalic contraction is definite and severe and is felt in the epigastrium. When rupture occurs the epigastric pain diminishes and the local pelvic peritonitis results on the right side of the pelvis or at the bottom of the pelvis cul de sac. This is usually unaccompanied by rigidity of the lower abdominal wall. The patient may seem better, the symptoms in the upper abdomen have diminished, there are no signs of rigidity of the abdominal muscles and very little indication of trouble in the pelvis. If the history of the pre-rupture symptoms are missed, it may be very difficult to come to the true realization of the cause of the patient's fever. Irritation of the pelvic wall may result in inflammation covering the obturator internus and rotation of the flexed thigh may elicit pain. Irritation of the bladder and rectum may be present. A mass may be palpable through the rectum. In this type of appendicitis the finger in the rectum is as important as the thermometer in the mouth. Intestinal obstruction is especially apt to occur in complicated cases of pelvic peritonitis.

The treatment of acute appendicitis is, as before, surgical but the treatment has not remained static. The principles of physiological chemistry apply here as well as in the patient with acute intestinal obstruction. The febrile patient with furred tongue and pinched facies, who is dehydrated and acidotic must have restoration of his fluid and electrolyte balance. Protein depletion must be recognized or anticipated. The use of amino acids have proven to be of great service in preventing the complications likely to arise in the presence of hypoproteinemia. The introduction of chemotherapeutic agents, particularly penicillin, allows for changes in surgical practice. The technical aspects of the appendectomy have not changed greatly although they vary. The various technics have their advocates because the surgeon may feel that one incision gives less exposure of the peritoneal contents or better exposure of the peritoneal contents, less chance of contamination, less shock, better drainage and better closure than another method.

The enthusiasm which accompanied the advent of penicillin was tempered somewhat in the minds of surgeons who found its limitations in abdominal conditions complicated by the presence of gram-negative bacilli, notably the Escherichia coli.

Penicillin in doses of 30,000 units every four hours intramuscularly appears to be slightly more effective than sulfonamides in controlling infections of appendiceal origin. In general, however, 30,000 units of

penicillin six times daily will not prevent the formation of intraperitoneal abscesses. The penicillin neutralizing function of the Escherichia coli can be overcome by high initial dosage according to Lieut. George Crile, Jr. and Captain James R. Fulton in a

between May 10, 1942, and August 18, 1946. (Table 1.) During this period there were 2,003 major surgical procedures, 616 or 30.7 per cent of which were appendectomies exclusive of incidental operations performed in conjunction with other pro-

TABLE II

PERFORATED APPENDECTOMY CASES REOPERATED

Name	Age	Symptoms Days before Admission	Reoperated Days after Appendectomy	Cases for Second Operation	Miller-Abbott Tube	Peni-cillin	Days of Dismissal after Appendectomy
L. J.	14	1 day	11 days	Intestinal obstruction	Yes	Yes	29 days
A. H.	15	2 days	13 days	Intestinal obstruction	Yes	Yes	28 days
J. S.*	13	1 day	7 days	Mesenteric thrombosis Intestinal obstruction	Yes	Yes	21 days
C. D.	46	2 days	13 days	Generalized peritonitis Dehiscence Abdominal wall	No	Yes	50 days
R. C.	37	5 days	3 days	Generalized peritonitis Dehiscence Abdominal wall	No	Yes	Death 7 days after appendectomy

* Rheumatic heart disease.

report of the U. S. Naval Medical Bulletin.[2] In vitro experiments indicate that the colon group of bacilli is not susceptible to treatment with penicillin but clinical evidence is accumulating that the larger doses are effective in controlling mixed infections. These authors recommend 100,-000 units given intramuscularly every two hours for two days and then 50,000 units every two hours for two days, followed by 50,000 units every four hours for two days and finally 25,000 units every four hours for two days, a total of 4,500,000 units in eight days. Lieut. Crile and Captain Fulton believe that a conservative attitude toward the complications of appendicitis has been responsible in no small part for the low mortality in their series of patients, and therefore, recommend conservatism and treatment with chemotherapeutic agents in late cases.

The effects of the use of penicillin are well brought out by the following survey of one surgical service* in our hospital consisting of operated cases of appendicitis

* The author's.

TABLE III

APPENDECTOMY DEATHS

Name	Sex	Age	Day of Symptoms before Admission	Day of Death	Cause	Peni-cillin
R. B.	Fe	45	3 days	2 days	Peritonitis	No
F. P.	Fe	45	several	3 days	Peritonitis	No
S. H.	Fe	60	7 days	2 days	Peritonitis	No
M. C.*	Fe	32	3 days	8 days	Pulmonary embolism	No
E. C.*	M	62	4 days	14 days	Pulmonary embolism	No
B. T.†	M	76	several	9 days	Pulmonary embolism	No
M. S.	Fe	60	5 days	7 days	Peritonitis	Yes
L. T.	M	68	3 days	12 days	Peritonitis Uremia Nephro-sclerosis	Yes
G. W.	Fe	5	several	2 days	Peritonitis	No
P. W.	Fe	11	5 days	35 days	Peritonitis	Yes
R. C.	M	37	5 days	7 days	Peritonitis Chronic alcoholism Dehiscence Abdominal wall	Yes

* All cases of death from pulmonary embolism were improving, had the drains removed and were afebrile. Embolism probably arose from a site of phlebothrombosis.
† Gallstone impacted in appendix.

cedures. They were classified as follows: acute 278; perforated 105; abscessed 13; subacute 168; catarrhal 4; mechanical 4 and

recurrent 44. It may be seen from the table that 18.6 per cent of the acute cases were of a perforated nature. Of the 616 patients of all types during the entire period there was a mortality rate of 1.7 per cent. In the period before June 15, 1944, 301 patients of all types carried a mortality of 2.3 per cent and after penicillin was introduced into this hospital the series of 315 patients of all types carried a mortality of 1.2 per cent. This represented a decrease in mortality after penicillin of 48 per cent. (Tables II and III.)

In those patients with perforation and frank abscess formation, 118 patients for the entire period of the survey carried a mortality of 9.3 per cent. Broken down into patients before penicillin was used, 40 of these patients with perforation and those with frank abscesses carried a mortality of 17.5 per cent. These same patients after penicillin, seventy-eight in number, carried a mortality of 5.1 per cent. The percentage decrease in mortality after penicillin was 70.9 per cent.

Five patients with perforated appendicitis and peritonitis were reoperated upon, two because of intestinal obstruction one because of mesenteric thrombosis with intestinal obstruction and two because of dehiscence of the abdominal wall. Resection of the terminal ileum was necessary in the one patient with mesenteric thrombosis. The Miller-Abbott tube for small intestinal intubation has proven of great value in instances of paralytic and mechanical ileus.

Of the deaths following appendectomy, six were caused by peritonitis; one by peritonitis with nephrosclerosis and uremia; one by peritonitis with chronic alcoholism and three were due to pulmonary embolism. All the patients who died from pulmonary embolism were improving at the time and had had the drains removed. They were afebrile. The embolism probably arose from a site of phlebothrombosis. It was discovered that one of these patients had a gallstone impacted in the appendix.

The clinical effects of penicillin in mixed infections is, therefore, striking and with the larger doses suggested by Lieut. George Crile, Jr. and Captain James Fulton, greater efficacy may be expected in overcoming the penicillin neutralizing function of the Escherichia coli.

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BEADED WIRE IN TREATMENT OF SLIPPED EPIPHYSIS OF THE HEAD OF THE FEMUR*

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THERE has been a multiplicity of treatments, operative and conservative, for slipped epiphysis of the head of the femur. Treatment and prognosis have generally varied according to the stage of the condition. In the preslipping stage results have generally been excellent with either conservative or operative treatments. Conservative treatment includes simple bed rest or traction; the operative treatment includes simple surgical procedures such as the drilling operation or the various methods of internal fixation through a lateral approach which avoids the hip joint.

In the late stage when not only has considerable slipping occurred but where the epiphysis has also fused in the deformed position, the accepted treatment is osteotomy, through the healed epiphyseal line, femoral neck or the subtrochanteric region. The former is generally avoided because of the danger of later degenerative changes of the femoral head and articular cartilage.

It is in patients in whom appreciable slipping has occurred and the epiphyseal line is still not closed that most of the difference of opinion exists and in whom many bad late results have occurred. Forcible manipulation and open reduction have given a high incidence of degenerative arthritis after several years whether or not aseptic necrosis had occurred. More recently Howorth and others have advocated a period of waiting either with bed rest and traction or internal fixation until the epiphyseal line has closed and then performing the corrective osteotomy. Results in these patients appear to have shown a distinct improvement over previous ones.

Another treatment in this group of sub-jects which would seem to offer a method of reduction with less risk is traction. Watson-Jones has recommended a period of ten days to two weeks in heavy traction and Wardle has devised a form of skin traction which allows for a pull in the direction of internal rotation of the hip with the hip being maintained in adduction. Wardle's results in a series of nine patients appear promising.

Skeletal traction allows a much greater pull and would seem to be especially applicable here. The purpose of this article is to present a form of skeletal traction by means of beaded wire through the lower femur, allowing for a strong, continuous pull in the long axis of the femur with an additional pull in the direction of internal rotation. The beaded wire, a Kirschner wire with a metal bead brazed to it, was devised by Thomson and Ferciot for use in fixation of oblique and spiral fractures of the tibia, the wires being incorporated in a cast. Briggs and Keats applied the wire for continuous traction in extracapsular fractures of the hip utilizing the traction in the line of internal rotation to good effect. Our use of beaded wire in slipped epiphysis was directly suggested by this work. The technic of application is simple The wire is applied under sterile conditions with local anesthesia three quarters of an inch above the medial condyle of the femur with a cannulated hand drill. The approach is from the medial side. A small skin incision is made. The wire is introduced until the bead is felt to rest against the femur; i.e., it cannot be pushed further. The bow is then applied and the traction cord attached. A second cord is then attached to the lateral portion of the bow

FIG. 1. Case I. A, anteroposterior view of left hip on admission showing characteristic displacement of capital epiphysis. B, lateral view of left hip taken eleven days after traction was instituted and six days after gentle manipulation showing slight posterior displacement of the epiphysis.

FIG. 2. Case I. A, anteroposterior view of left hip after fifteen days of traction showing complete reduction. B, lateral view of left hip fifteen days after commencement of skeletal traction.

FIG. 3. Case 1. A, anteroposterior view of the left hip showing Moore pins *in situ* with almost complete closure of the epiphyseal line, two and one-half months following insertion of Moore pins and three and one-half months following start of traction. B, anteroposterior view of the left hip taken June 10, 1946, showing complete fusion of epiphyseal line.

where it grips the wire, the pull being made perpendicular to the femur in an upward direction. This applies force in the direction of internal rotation and the bead prevents slipping of the wire.

We have had four experiences in the use of this technic, as described in the following case reports.

CASE REPORTS

CASE 1. A fifteen year old boy was admitted on May 16, 1945, to the New Jersey Orthopaedic Hospital complaining of pain in the left thigh. In October, 1944, the patient began to experience pain in the left thigh, and walked with a limp. The limp improved after about three months for a short time but then recurred with pain. Pain occurred in the left anterior and medial thigh and was referred down to the knee. There were no constitutional symptoms. His personal and family history were irrelevant.

On examination his general appearance was that of a rather thin boy appearing about fourteen years of age, not looking acurely or

FIG. 4. Case 1. Lateral view of the left hip taken June 10, 1946, showing complete fusion of the epiphyseal line.

chronically ill. The left lower extremity was held in about 40 degrees external rotation, as the patient lay in bed. Passive flexion of the left hip lacked 10 degrees of a right angle.

FIG. 5. Case II. Anterior view of hips showing marked displacement of right femoral epiphysis.

External rotation, passively, was unusually good; internal rotation, however, failed even to reach the neutral point by 10 degrees. Abduction was almost normal. There was no tenderness in the region of the hip or thigh on the left side. There was ½ inch shortening on the left side as measured from the anterior superior iliac spine to the tip of the medial malleolus.

X-ray of the left hip showed characteristic appearance of slipped epiphysis with downward and posterior displacement of the epiphysis.

The following day a beaded wire was inserted in the supracondylar region under local anesthesia with sterile precautions. Following this, traction was made on the spreader. After five days the left hip was gently manipulated, under sodium pentathol anesthesia, after which x-ray showed an apparent partial correction. X-ray taken June 2, 1945, showed apparently perfect correction in both views. Initially he had 20 pounds of direct pull and 10 pounds in the direction of internal rotation, which in a few days was increased to 15 pounds in the direction of internal rotation. Because of his lack of weight (97 pounds) his left buttock was pulled off the bed and for about a week, counter weights were required to hold down his pelvis. Following x-ray on June 2, 1945, direct pull was reduced to 15 pounds and internal rotation was reduced to 10 pounds. The wire was removed on June 8, 1945, after sixteen days because of local infection. On removal of the wire a few drops of thick pus exuded. Wet dressings were applied and penicillin given. The temperature rapidly returned to normal and the infection cleared up completely.

The patient was placed in Russel traction on June 12, 1945. After having been out of traction completely for four days, on June 18, 1945, through a lateral incision three Moore pins were inserted into the head of the femur. Postoperative course was uneventful. The patient
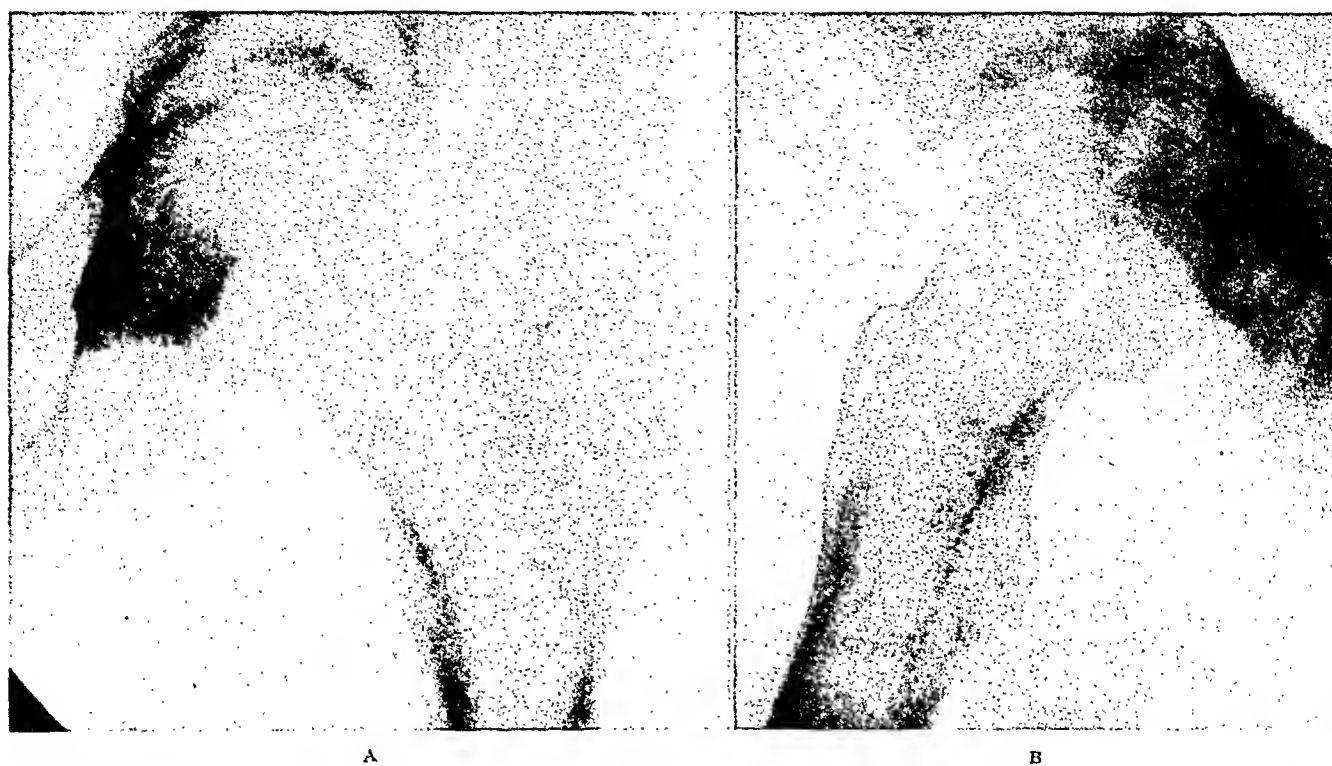
FIG. 6. Case II. A, anteroposterior view of right hip June 4, 1945, following drilling of hip showing complete reduction. B, lateral view of the right hip June 4, 1945, showing complete reduction.
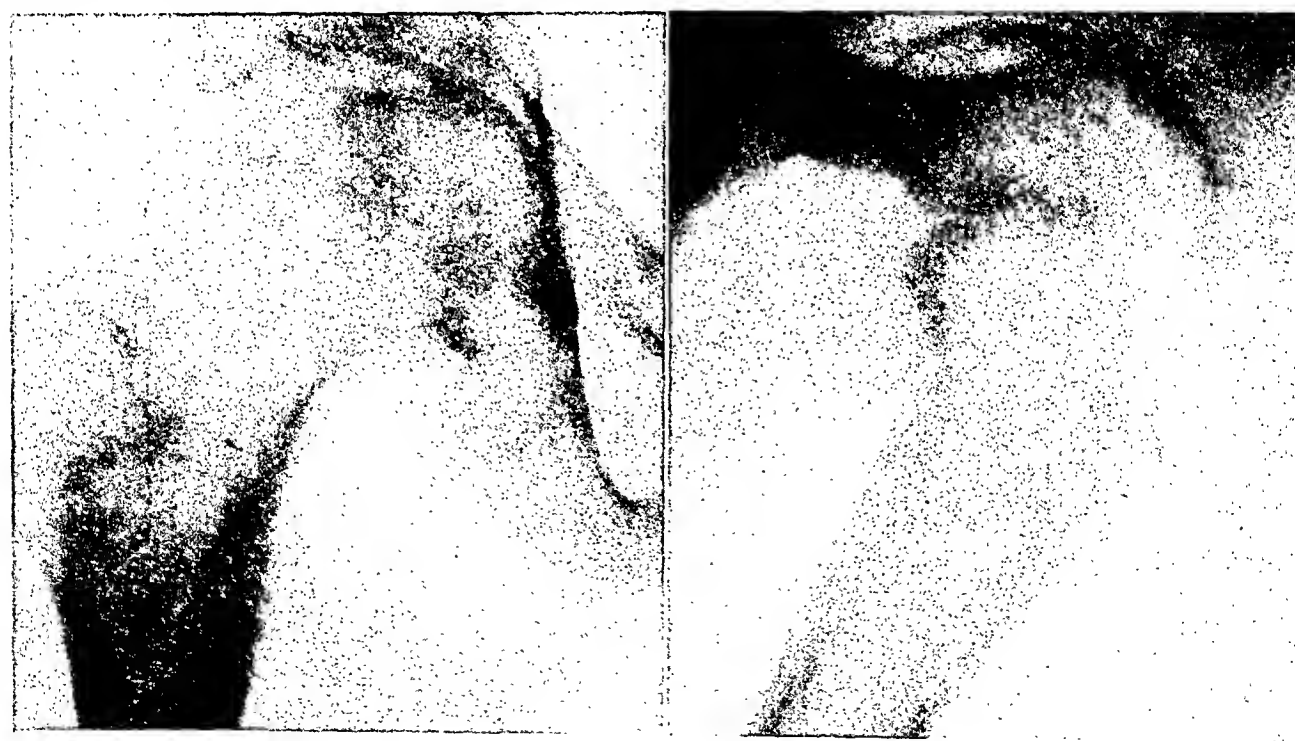
FIG. 7. Case II. A, anteroposterior view of right hip four months later showing complete reduction and beginning disappearance of the epiphyseal line. B, lateral view of right hip four months after admission showing complete reduction.

was discharged on July 5, 1945, in good condition. He was up and about on crutches. Anteroposterior and lateral views of the left hip in the x-ray taken September 5, 1945, showed the three Moore pins in good position.

The head was in excellent relationship to the neck. There was no evidence of avascular necrosis; the epiphyseal line showed beginning fusion. External rotation was good; internal rotation to 10 degrees past the neutral point.

A   B

FIG. 8. Case II. A, anteroposterior view of the right hip taken June 10, 1946, showing complete fusion of the epiphyseal line. B, lateral view of the right hip taken June 10, 1946, showing complete fusion of the epiphyseal line.

There was no pain; leg lengths were equal. On January 4, 1946, x-ray showed almost complete obliteration of the epiphyseal line. There was no evidence of bony reaction about the Moore pins. The joint space was well preserved.

When last seen on June 10, 1946, the gait was normal. There was no pain. The patient enjoyed full activity. X-ray showed almost complete closure of the epiphyseal line. The head and joint appeared normal.

CASE II. The patient, a sixteen year old boy, was admitted on May 1, 1945, complaining of pain in the right knee, of two weeks duration. About a year before the patient complained of pain in the right knee and had a limp for about two months. This cleared up until about two weeks before admission when the patient again complained of pain in the region of the right knee and began to limp. No constitutional symptoms were presented on either occasion. Personal and family history were irrelevant.

On examination, the general appearance was that of an obese, white boy of sixteen to seventeen years of age, rather pale, not appearing acutely or chronically ill, sitting in a chair in much apparent discomfort. As the patient lay in bed the right lower extremity lay in external rotation. All motions of the hip were restricted. However, internal rotation was not possible even to the neutral point. Abduction was also markedly restricted. Motion of the right hip caused pain referred to the inner aspect of the lower thigh in the knee region. There was shortening of ½ inch on the right.

X-ray of the right hip showed an extreme displacement of the femoral capital epiphysis posteriorly and downward.

The patient was placed in Russel traction to the right lower extremity for six days, after which time x-ray of the hip showed slight, if any, correction of the slipping. The following day, under sodium pentathol anesthesia, the right hip was gently manipulated with traction and internal rotation without apparent correction. A beaded wire was then inserted under sterile conditions into the right femur above the condyles, from medial to lateral. Traction was made postoperatively on the beaded wire as described. Twenty-nine pounds of direct traction was made with 15 to 20 pounds of pull in the direction of internal rotation. X-rays taken five to eight days later showed a partial correction of the slipping. After ten days the right hip was opened through a Smith-Petersen incision and the relations within the acetabulum appeared normal. Check-up x-rays, on the operating table, showed complete correction of the deformity. The incision was closed and a

lateral incision was made, through which multiple drillings were made into the head and neck of the femur. Postoperatively, the patient ran a low grade fever.

The patient was put back in traction on the beaded wire with 20 pounds direct pull and 10 pounds in the direction of internal rotation. After one week this was further reduced to 15 pounds of direct pull and 8 pounds in the direction of internal rotation. After another week it was further reduced to 10 pounds direct pull and 5 pounds in the direction of internal rotation. He was discharged from the hospital on July 5, 1945, in good condition, on crutches. On August 6, 1945, he returned for examination. He complained of no pain, incisions were well healed. He was getting along well on crutches. Flexion of the right thigh on abduction to 90 degrees was normal. Internal rotation of 20 degrees was present. External rotation was normal. X-rays taken September 5, 1945, showed beginning closure of the epiphyseal line with the head in excellent relationship to the neck. On October 2, 1945, the patient had almost full range of motion on the affected side in abduction, flexion and both rotations. Leg lengths were the same. No pain was present and the patient was walking without complaint.

He was last seen on June 10, 1946. He had no complaints. There was no limp. Leg lengths were equal. Motion of the right hip was normal as compared with the left. X-ray showed closure of the epiphyseal line. Head of the femur and hip joint appeared normal.

CASE III. The patient, a fourteen year old white male, was admitted complaining of pain in the right hip for six weeks, increased for two weeks. He had had a slipped epiphysis on the left hip three years before.

On examination, the patient walked with a right sided limp holding the right lower extremity in external rotation. The right hip showed flexion to 80 degrees. Internal rotation lacked 20 degrees of neutral. X-ray showed marked backward and downward displacement of the right upper femoral epiphysis.

On May 29, 1946, a beaded wire was inserted into the right femur. The application was unsatisfactory and on June 14, 1946, it was removed and another wire applied. Direct traction of 25 pounds was applied with 10 pounds in the direction of internal rotation.

An x-ray taken July 8, 1946, showed no change in position.

It was then decided to postpone any surgical procedure and to have the patient rest at home. Later osteotomy was contemplated.

CASE IV. The patient, a twelve year old white male, was admitted on May 25, 1946. For several months he had reported an occasional sticking pain in the left hip when he walked or ran. Pain disappeared when he sat down or rested. He had pursued his usual activities until three weeks before admission when he developed pain in the left hip at rest.

On examination the patient appeared a rather obese twelve year old boy, eunuchoid, with heavy trunk and poor muscular development. He walked with his left lower extremity in marked external rotation.

As he lay in bed the lateral border of the left foot lay flat on the bed. Internal rotation lacked 10 degrees of the neutral position. There was moderate restriction of abduction and slight restriction of adduction. There was no inequality of leg lengths.

X-ray showed a moderate amount of slipping of the left upper femoral epiphysis.

On May 27, 1946, a beaded wire was inserted into the left femur as described. Traction was gradually increased so that at the end of a week 30 pounds of direct traction and 13 pounds of traction in the direction of internal rotation were being applied. X-ray taken June 8, 1946, showed complete correction. On June 10, 1946, three Moore pins were inserted. The postoperative course was uneventful.

When last seen on November 12, 1947, the patient was symptom-free and there was no restriction of motion on physical examination.

CONCLUSION*

A method of treating some cases of slipped femoral capital epiphysis with the beaded wire has been presented.

Elapsed time is too short to evaluate the end results. However, the excellent correction in three of the four patients and the very good functional result to date in these three patients after a year justify further use of this procedure. The risk involved is small in comparison with the possible benefits. In addition, in the event of failure to achieve correction, the hip itself has not

* Since this article was submitted, several more patients have been treated with this technic and these will be recorded in a subsequent article.

been compromised and an osteotomy later may still produce a good result.

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PATIENTS with fractures of the lower limb never should be transported until after splinting has been applied. Hospitalization is necessary for the modern treatment of fractures of the leg. Order X-ray examination before deciding upon the method of further treatment.

From "Fractures and Dislocations for Practitioners" by Edwin O. Geckeler (The Williams & Eilkins Company).

MODIFIED INDIRECT INGUINAL HERNIORRHAPHY

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THE diagnosis of an indirect inguinal hernia if it is complete and presents itself to the examiner as a mass either in the groin or scrotum is seldom a problem. However, if the condition is incomplete, a bubonocele, that is the protrusion exits through the internal abdominal ring and does not descend the entire length of the inguinal canal or there are no contents within the sac at the time of examination, the diagnosis may be a little more uncertain. When a patient maintains that there was a lump or a child's parent affirms that a mass has been seen one may accept it as presumptive evidence of the existence of a herniation. However, one cannot make such a diagnosis solely on the finding of a dilated or enlarged external inguinal ring.

Henning[5] states that all hernias are congenital in origin with the exception of the traumatic type which shows evidence of injury. This statement requires clarification as invariably the findings of trauma are not visible even at the operating table. It is the author's belief that the potentiality for herniation is congenital rather than profess the characterization that all hernias are congenital. The persistence of the processus vaginalis in itself does not always portend a hernia. It may be described as an enlage in which the final result of protrusion may not necessarily follow. One author voices the opinion that 4.4 per cent of male infants have hernias in the first year of their life; however, most of them disappear after a time.[10]

Erdman[3] maintains that the etiological condition of hernia can be due to one or to a combination of factors. They are a defective closure of the processus vaginalis following the descent of the testis through the canal, sudden increased intra-abdominal pressure and failure in the inguinal shutter mechanism. The latter fault is offered by Keith[10] as the cause of hernia and he further describes it as being formed by the conjoined internal oblique and transversus muscles. Under reflex-nervous stimuli these muscles act against the inguinal ligament serving to close the deep abdominal ring.

In the final analysis the causation of inguinal indirect hernia can be accepted to comprise either one or a multiplicity of factors. Beyond all these one must take into consideration the altered evolutionary dynamics of the human species due to their upright stature.

To understand the reasons for the methods employed in the repair and the prevention of recurrences of oblique inguinal hernia a thorough knowledge of the inguinal region is a prerequisite. It is not within the scope of this paper to enter into a complete and detailed anatomic description. Reference should be made to any standard textbook on the subject; however, for the purpose of recapitulation and for some recent concepts the essential highlights are noted. (1) Only the medial portion of Poupart's ligament where it forms the lacunar or Gimbernat's ligament is attached to the pectineal line. Furthermore, Poupart's ligament's only connection to the fascia of the thigh is by a very thin investing fascia;[12] (2) the ligament of Sir Astley Cooper is attached to the pectineal line. With the subject in a standing position this ligament is almost horizontal in position; (3) the normal insertions of the internal oblique and transversus muscles are the pectineal line and not the inguinal ligament; (4) the internal abdominal ring through which the sac protrudes in order that a hernia can be termed as indirect, is situated in the transversalis fascia. Its medial boundary is the inferior epigastric vessels. This anatomic landmark is not a

ring but an hiatus and is funnel-shaped; (5) Hesselbach's triangle is located medially to the inferior epigastric vessels which form its lateral boundary. The other limits of this space are the lateral edge of the inguinal ligament; direct inguinal hernias make their appearance first through this area.

Recurrences of an oblique inguinal hernia are statistically reported to be between 2 and 20 per cent.[17] Consideration of recurrences due to surgical complications as infections and the disruption of the wound are omitted in this discussion. Heath, Brown and Saunders[7] in an analysis of nineteen recurrent hernias found sixteen in which the original sacs were inefficiently removed. This is gross surgical negligence wherein the basic principle of herniorrhaphy was completely ignored, therefore, recurrence should not rightfully be claimed; rather, no surgery for hernial repair has been undertaken. The incision may as well have been located somewhere else on the abdomen unrelated to the inguinal region.

An important cause for failure ascribed to by McVay and Anson[12] is the suturing of the internal oblique and transversus abdominal muscles to Poupart's ligament which is not the normal insertion for this musculature. The failure of the closing or the narrowing of the internal abdominal ring is believed to be another one of the important responsible factors in the production of many recurrences of the indirect variety. It can be assumed that if this hiatus can be completely obliterated, of course entailing the sacrifice of the spermatic cord, an indirect hernia will not recur. This, however, is not suggested for more than one obvious reason.

The surgical procedures which are offered and continue to be offered are legion.[2,4,9,14,15] It is unnecessary to enumerate all of them. The lack of one universal method is due not only to individual preferences but also to the constant search for a repair which will result in a minimal number of failures. For this reason the closest cooperation between surgeon and anatomist is required.

The laparotomy approach by La Rocque and Banergie is cited by Erdman.[3] The sac is dissected and ligated intraperitoneally. Likewise, a similar attack through a midline infra-umbilical incision is fostered by Jennings, Anson and Wright.[9] The hernial sac is dissected retroperitoneally with a high ligation and the internal hiatus is closed by approximation of the transversalis fascia over the opening.

As one peruses the literature on the subject he becomes amazed and arrives at the conclusion that the last word has not been written or said. Furthermore, the impression is gained that all procedures are modifications with attempted improvements of earlier basic herniorrhaphies, such as that of Bassini, Halsted and Wylly Andrews.

Although fundamentally the high ligation and obliteration of the sac are the cardinal therapeutic tenets, most men implement the procedure with technics either striving to close the deep abdominal hiatus or strengthening the floor of inguinal canal. Harkins et al.,[5] after the usual sac ligation, purse-string the transversalis fascia around the spermatic cord. This they follow by suturing the conjoined tendon after the McVay[12] method to Cooper's ligament adjacent to the anterior ramus of the pubis up to the femoral vessels which are about 5 cm. lateral to the pubic spine; the first suture being placed 4 cm. from that point. They vary the procedure of transplanting the cord over the aponeurosis as in the original Halsted operation or follow the typical Bassini operation by leaving the cord in the space beneath the aponeurosis.

Ligation of the sac with suture of its stump beneath the conjoined tendon and tied to the fascial part of the internal oblique muscle is employed by Henning.[8] To augment or fortify the floor of the canal he resorts to a modification of the Andrews' operation. Reliance is placed on a series of special non-absorbable sutures which start

by piercing the lateral leaf of the external oblique aponeurosis as near the symphysis pubis as possible. They then pass under Poupart's ligament across the bottom of the wound and pick up the conjoined tendon and back through Poupart's ligament. Then they traverse the medial leaf of the external oblique fascia and finally pass again through the lateral leaf near the point of commencement. This is followed by fascial imbrication.

Moschowitz[15] in the repair of the femoral hernia makes use of Cooper's ligament as the main binding post. Since then, others[1,12,14,16] are employing this ligament in the repair of inguinal hernia. Mastin[11] in an effort to further barricade the deep abdominal hiatus obliquely incises the internal oblique muscle close to its attachment to the aponeurosis and places the cord through it.

The details of the described operation does not claim entire originality, but rather, combines parts of different procedures which in the author's hands have proved highly satisfactory. It is fairly well accepted that direct and indirect inguinal hernias are two different entities having different dynamics and therapeutic needs. Yet, a patient who has had an indirect herniorrhaphy consisting of a simple ligation and excision of the sac develops a direct hernia—to him the original operation is a failure and he cannot be influenced to view it otherwise. Therefore, the technic presented buttresses the area of Hesselbach's triangle and, in addition, forms another barrier at the internal abdominal ring.

TECHNIC

1. Incision beginning 2.5 cm. below and medial to the anterior superior iliac spine continues parallel from Poupart's ligament to approximately 2.5 cm. proximal to the symphysis pubis.

2. With a finger inserted through the superficial or external ring, the aponeurosis of the external oblique is divided through it and extended laterally towards the anterior superior iliac spine.

3. The inner aspect of the inguinal ligament is cleared with a piece of gauze and Cooper's ligament is isolated and freed of connective tissue.

4. The cord is displaced from the canal, the indirect sac identified and dissected free from the cord structures and followed up as high as possible to the glistening peritoneum.

5. The sac is ligated at its base and the excess removed.

6. With the cord still retracted, the transversalis fascia is plicated and employing the fascia the deep hiatus is made snug around the cord. If the inferior epigastric vessels interfere they are severed and carefully ligated.

7. The conjoined tendon is sutured to Cooper's ligament (absorbable or nonabsorbable material may be used). The first suture includes the tendon and passes through the periosteum overlying the pubic spine and the lacunar ligament. The second suture starts in the conjoined tendon opposite the site of the femoral vessels and with the index finger protecting the vessels the needle impinges Cooper's ligament. Serial sutures, as necessary, are placed between the first and second sutures.

8. A cross or perpendicular (to the fibers) incision is made through the internal oblique muscle at the level of the narrowed deep inguinal ring for approximately 2 cm. The cord is then set in this incised defect and the lateral edges of the cut muscle are re-apposed.

9. Complete transplantation of the cord is accomplished by attaching the medial leaf of the aponeurosis of the external oblique to the shelving edge of Poupart's ligament. The lateral leaf is then imbricated over the medial leaf and the cord is finally replaced on the newly created floor. Or, on the other hand, the cord can be transplanted after performing Step Eight by placing it in the recess and the divided aponeurosis of the external oblique resu-

tured, as in the Bassini operation over the cord.

10. The skin is closed in the usual manner.

COMMENTS

Thirty-eight cases of oblique hernia have been done employing the above technic.

TABLE I

CASES PERFORMED BY DESCRIBED METHOD

	No. Cases	Six to Twelve Mo. Follow-up	Complications	Failures
Non-absorbable material, cord above aponeurosis	16	12	One wound broke down	0
Absorbable material, cord above aponeurosis.......	18	10	none	0
Absorbable material, cord beneath aponeurosis.....	4	2	none	0

(Table I.) Of these, twenty-four have been followed for at least six to twelve postoperative months. In one patient the wound broke down and the non-absorbable material which was used was picked from the wound for quite a period of time before complete healing ensued. Nevertheless, the result in this patient was good. Thus far there have been no evidence of recurrence in any of the subjects.

SUMMARY

1. The etiological condition or the origin of the oblique or indirect inguinal hernia is ascribed to certain factors; the persistence of the processus vaginalis, a sudden increase of intraabdominal pressure or the failure of the inguinal shutter mechanism.

2. Some of the causes for failure or recurrence are cited. Either the sac is not completely or satisfactorily ligated, the deep inguinal hiatus is not repaired or Cooper's ligament is not employed as the anatomic insertion of the internal oblique and transversus muscles.

3. Several types of surgical procedures are discussed as to their major purposes.

4. A modified technic is described to help obliterate the deep hiatus and fortify the triangle of Hesselback.

5. Thirty-eight cases were performed using this procedure. Although the follow-up period is admittedly short for complete evaluation, the results have been uniformly satisfactory to warrant the continuation of this method.

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NEW TEST IN DIAGNOSIS AND SURGICAL TREATMENT OF VARICOSE VEINS*

TWO HUNDRED VEIN LIGATIONS EVALUATED

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THE modern treatment for primary varicose veins of the long saphenous system with their incompetent valves has been ligation and resection at the japhenofemoral junction followed by insection of sclerosing solution in the varices. The literature is replete with articles stressing the importance of ligating all branches in the region of the fossa ovalis. There has not been the same unanimity of opinion regarding the importance of ligating all incompetent communicating veins or "blowouts."

Some writers minimize the importance of incompetent communicating veins. Heyerdale and Anderson[6] say that these veins are relatively uncommon and think that the rapid filling of superficial veins in the ordinary Trendelenburg Test is caused by a reflux from the lesser saphenous vein and not from incompetent communication veins. Luke,[9] on the basis of venography studies, claims that incompetent communicating veins are very rare and unimportant. Other very competent investigators, notably Pratt,[15] Mahoner and Ochsner[12] and Linton hold quite the opposite views, as do most specialists in this field. Dean and Dulin[3] list them as the primary cause of recurrence.

Our experience has proved that "blowouts" are the chief cause for recurrence of varicosities. We found that whenever a "blowout" was overlooked, injections of sclerosing solution would not obliterate the vein fed by the perforator. Back flow through the incompetent perforator either prevented a firm sclerosis or caused recanalization.

After Mahoner and Ochsner[11] published the account of their comparative tourniquet test in 1936 we used it to advantage to discover "blowouts." The comparative tourniquet test depends upon emptying of the superficial veins through the deep system (modified Perthes test). It only indicates the segment of the leg or thigh involved. For this reason others have introduced modifications of the standard Trendelenburg test to locate more accurately the site of "blowouts." Pratt's[14] two tourniquet technic introduced in 1939, was the first of these. McCutchen,[13] Lyall[10] and others have used two tourniquet modified Trendelenburg tests for the same purpose. All such tests depend upon trapping the "blowouts" and must be repeated a number of times.

Seeking a more accurate and less time-consuming method to locate incompetent communicating veins, Dr. Don McLean of our clinic suggested a modification of the Brodie-Trendelenburg test. In a simple manner this test simultaneously determines both the presence and site of a "blowout" and the type of Trendelenburg reaction. We have named this the "multiple tourniquet test." A search of the literature fails to reveal any other report of a multiple tourniquet test.

The multiple tourniquet test was mentioned by the author in 1940,[18] but not stressed, as we had insufficient data to substantiate its value. We have used it since 1939 and now have ample clinical proof of its usefulness.

MULTIPLE TOURNIQUET TEST

This test is done as follows: The patient lies on the examining table with the lower

extremity elevated until the superficial veins are emptied. Tourniquets are placed about the upper, middle and lower thigh and at least one below the knee. When the patient stands the tourniquets are removed from below upward within thirty-five seconds. A rapid filling of any segment before the highest tourniquet is removed indicates a Trendelenburg double positive reaction. The segment is noted and the test repeated observing the exact location of the "blowout." This is marked with a silver nitrate stick. Such marking remains visible for several weeks and will not wash off when the site is prepared for surgery. In performing this test the leg is observed both anteriorly and posteriorly. Thus, the possibility of an incompetent lesser saphenous vein is explored.

With this test "blowouts" are easier to locate than with the comparative tourniquet test because the rapid filling of a vein is more obvious than its failure to empty. Moreover, many "blowouts" which would empty with the comparative tourniquet test will fill when the multiple tourniquet technic is followed.

Pratt[15] remarks that the comparative tourniquet test is difficult to perform and to interpret and is so time-consuming that it is not useful in the clinic. We believe that all two tourniquet tests are also time consuming and lack the degree of accuracy we have obtained with the multiple tourniquet technic. We frequently locate several "blowouts" in the same extremity. Of 102 high and low ligations there were seventeen with two and four with three or more ligations on the same extremity.

Ochsner[12] advises, when incompetent communicating veins are found with his comparative tourniquet test, that the saphenous vein be ligated at knee level. He admits that such ligations "do not preclude the possibility of recurrences through collateral channels around the lowest transection and ligation." We believe that recurrences can be avoided by ligating all "blowouts" at their site. The finding of only 3.4 per cent recurrence

where this procedure has been followed is proof of its value.

The multiple tourniquet test is not only accurate but simple to perform. Rarely is it necessary to repeat it more than once. It is time-saving because not only is the

TABLE I
TYPES OF SAPHENOUS VEIN LIGATIONS

Total Liga-tions	High Liga-tions	High and Low Liga-tions	High and Lesser Saphe-nous Liga-tions	Low Liga-tions	Lesser Saphe-nous Liga-tions
200	60	102	13	20	5
Per cent	30	51	6.5	10	2.5

Trendelenburg reaction indicated but also, in double positive cases, the "blowout" is accurately located. Therefore, it readily lends itself to use in a busy clinic.

EVALUATION OF 200 LIGATIONS

Two hundred consecutive ligations on 140 patients, all tested with the multiple tourniquet technic, were selected for analysis.

Table I shows the distribution of the various ligations. It is significant that in this series 57.5 per cent of all operations were combined high and low or high and lesser saphenous vein ligations. In a report made by the author in 1940[18] on a series of 100 vein resections, I had only 21 per cent of multiple ligations. In this earlier series when only the classical Trendelenburg test and the comparative tourniquet test were used, 64 per cent of all operations were high saphenous resections only.

Table II indicates the value of the multiple tourniquet test in locating "blowouts." By comparison with the previous series it will be noted that both the positive and negative Trendelenburg tests were almost twice as frequent in the former series whereas in the present series the Trendelenburg double positive occurred two and one

half times as often as in the earlier group of cases. Our findings, therefore, are quite the opposite of Heyerdale and Anderson who claim that incompetent communicating veins are relatively uncommon. Since the same group of surgeons were responsi-

TABLE II
VALUE OF MULTIPLE TOURNIQUET TEST IN LOCATING INCOMPETENT COMMUNICATING VEINS

Trendelenburg Tests			
	Positive (per cent)	Double (per cent)	Negative (per cent)
Series of 100 cases using classical Trendelenburg test...........	58	21	21
Present series 200 cases using multiple tourniquet test...........	30	57.5	12.5

ble for both series of cases referred to above and since the only difference in diagnostic technic is the use of the multiple tourniquet test on patients in the present series, it seems obvious that this test is largely responsible for such a significant increase in the incidence of Trendelenburg double positive cases.

FREQUENCY OF RECURRENCE OF VARICOSITIES

When the ligation and injection method of treatment has been employed for varicose veins the frequency of recurrence has been variously estimated at from 10 to 25 per cent within one year in even the most favorably treated patients, according to Dean and Dulin.[3] Sarma[17] reported recurrences up to 1930 of 16 per cent but by adopting additional ligation in the lower part of the thigh whenever indicated, the number of failures dropped to between 6 and 7 per cent.

Table III gives the per cent of recurrence following the various types of ligations. The overall recurrence rate of 5.5 per cent cuts in half the 11 per cent for the series reported by the author in 1940.[18] In this

previous group of patients there was a recurrence rate of 16.7 per cent where high ligations alone were done. To me this means that previously we were missing "blowouts" in one out of every six patients. The low incidence of 3.4 per cent recurrence

TABLE III
FREQUENCY OF RECURRENCE OF VARICOSITIES

	Following all Ligations	Following High Ligations	Following High and Low Ligations Including Those in Which Lesser Saphenous was also Ligated	Following Low Ligations	Following Lesser Saphenous Ligations
No. of operations......	200	60	115	20	5
Recurrences.	11	4	4	2	1
Per cent of recurrences	5.5	6.6	3.4	10	20

where multiple ligations were performed speaks well, I believe, for the value of the multiple tourniquet test as a real diagnostic aid. This is especially so when it is recalled that the Trendelenburg double case has been so prone to develop new "blowouts" after surgery.

Of the twenty low ligations reported, all but five were recurrences following high ligations not included in this series. Most of the high ligations had been done elsewhere. The five low ligations were undertaken with the realization that recurrences were likely. But either because the varicosities were mild or because of concurrent diabetes or cardiac disease the lesser surgical procedures was elected. The two recurrences reported were in this small group of five patients. Mahoner and Ochsner[11] remark that low ligation in many cases gives the best immediate results. But they warn that one must interrupt the saphenous vein above its tributaries if recurrence is to be avoided.

The lesser saphenous vein was encountered in 7 per cent of Larson and Smith's[8] series of 491 cases. White and associates[19] using a two tourniquet test, found 13 per cent of lesser saphenous veins involved in 214 patients. By our multiple tourniquet

TABLE IV
AGE INCIDENCE

Age in Years	Men	Women	Total	Per Cent of Total
0–20	0	1	1	0.7
21–30	2	5	7	5.1
31–40	7	19	26	18.7
41–50	11	17	28	20.
51–60	20	28	48	34.5
61–70	6	12	18	12.9
71–80	3	3	6	4.3
Over 80	5	0	5	3.6

test we found only 2.5 per cent of the lesser saphenous veins alone involved. By including those patients in whom the lesser saphenous vein was incompetent in the presence of an incompetent greater saphenous vein, the incidence rose to 9 per cent. We thoroughly agree with Larson and Smith when they report that the importance of the lesser saphenous as a factor of venous stasis has been underemphasized. We cannot agree with them in their belief that ligation of the lesser saphenous vein will make ligation of "blowouts" unnecessary. The data presented in this report should sufficiently emphasize the value of ligating all "blowouts" encountered.

Table IV gives the age incidence in this series. Our findings of 73.2 per cent of patients between the ages of thirty and sixty years parallels the 75 per cent incidence for the same age group as reported by Hawkes and Hewson[4] for 600 subjects. Their ratio of women to men was three to one. Ours was eight to five. Our youngest patient was a girl of sixteen years and the oldest a man more than eighty-three years old. We did vein ligations on eight people over seventy, five of whom were eighty or more years old. Results in these patients were excellent. None of them has had a recurrence. They are all much more comfortable. We believe that age in itself is not a contraindication to vein surgery. We see no reason to deny the relief afforded by such surgery to the older age group.

RETROGRADE INJECTIONS ABANDONED

Since the technic of the high saphenous ligation operation is now quite well standardized, it seems superfluous to repeat it here. However, retrograde injection of sclerosing solution at the time of operation, advocated by the Mayo clinic group,[1] has been widely accepted as an essential part of the operative technic. We used it routinely until 1939 when we abandoned it except in a few individuals where large varicosities are present in the upper thigh. In our opinion, it is a procedure fraught with definite danger and many disadvantages. Homans[7] has warned of the danger of thrombosis and embolism following retrograde injections, particularly low in the saphenous system. Atlas[2] reports five patients with deep thrombosis following retrograde injections. Two patients died of pulmonary embolism. Aside from these real dangers there are many disadvantages, chief of which are: (1) the chemical phlebitis produced causes the patient to go to bed, thus defeating the ambulatory nature of the operation; (2) it is ineffective unless large amounts of sclerosing solution are used; (3) if several weeks are allowed for the veins to collapse, less solution will be needed for sclerosis. Harkins and Schug[5] and others have stressed similar reasons for abandonment of retrograde injections. We believe that the ambulatory feature of vein ligations is so important that anything that interferes with it, including retrograde injections, should be sacrificed to keep the patient on his feet. Since we ceased using retrograde injections we have been able to get our patients back to work within forty-eight to seventy-two hours. This is an economic advantage so important to many patients that they do not mind a few additional visits for sclerosing injections.

LOW LIGATION TECHNIC

Although the literature and text books adequately cover the technic of high saphenous vein ligation, little is said regarding low ligation. It is true that they are simple operations. However, because the vein is directly under the skin, care must be exercised not to nick the vein. The incision should be into but not through the skin. The final opening is made by spreading the skin apart with a small hemostat. The use of Allis clamps on the skin is to be condemned because the trauma thus produced causes the area to become devitalized and frequently causes slough. Traction sutures of nylon are recommended for retraction. The vein must be dissected one-half inch each way from the incisional margin. We have experienced delayed healing and stitch infection in low ligation wounds due to the use of catgut, silk and nylon. We have had the least reaction when we used No. 30 cotton for ties. Closure with a subcuticular suture of No. 00 nylon leaves a fine scar hardly visible after six months.

FOLLOW-UP

Adequate sclerosis of any varicosities remaining after ligation is essential to complete treatment. The patient should be observed three, six and twelve months after the last course of injections. Quite frequently, a few small varices will be found which were not present during the course of intensive treatment. Unless they are also sclerosed, they may be the site of subsequent "blowouts."

In our experience sodium morrhuate has been the best sclerosing agent. Soricin is nearly as good and is used whenever a patient exhibits a sensitivity to sodium morrhuate. We have found that up to 4 cc. of sclerosing solution is a safe amount to inject at one time if spread over two or more segments of the vein. By applying an elastic bandage to the leg for the first twenty-four hours after injections are given, we find that less solution is needed and disfiguring knots in the leg are avoided.

Linton advocates vein stripping rather than individual ligation of incompetent communicating veins. I believe that vein stripping is an unnecessary procedure in all but the exceptional case. If the incompetent perforators are properly ligated, the reverse flow of blood will be prevented. Vein stripping cannot do any more than this. But it is a much more formidable operation which carries with it a definitely high morbidity. Even when three or four "blowouts" must be ligated the patient remains ambulatory. He is definitely a bed patient following vein stripping. Above all, we cannot see why the rather formidable operation of vein stripping should be done routinely when we are able to effect 96.6 per cent cures with the less formidable high and low ligation technic which permits our patients to return to work in two or three days.

SUMMARY

1. A new diagnostic test, called the multiple tourniquet test is presented to locate accurately incompetent perforator veins in the thigh and leg in Trendelenburg double positive cases.

2. With this test both the regular Trendelenburg determination and the location of "blowouts" are simultaneously accomplished. Because the test is simple and quickly performed it lends itself to clinic use.

3. Two hundred consecutive ligations on 140 patients were analysed to demonstrate the value of this test. It is a significant fact that 57.5 per cent of all operations were combined high and low or high and lesser saphenous vein ligations as compared with only 21 per cent of multiple ligations in a previous series where this test was not used.

4. The recurrence rate of 3.4 per cent for multiple ligations was contrasted with a national average of from 10 to 25 per cent.

5. The importance of the lesser saphenous vein as a factor of venous stasis was emphasized and an incidence of 9 per cent reported.

6. Reasons why the author abandoned retrograde injection were discussed and

the danger of deep thrombosis and embolism stressed.

7. The author's technic for low ligations was described.

8. The importance of an adequate follow-up of all treated patients was emphasized.

9. Reasons why the author considers vein stripping operations unnecessary when incompetent perforators are encountered were presented.

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TREATMENT OF VARICOSE ULCER

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OF the many complications associated with varicose veins, ulcerations are the most important because of their frequent occurrence and the disability and suffering which they cause.

There is no standardized treatment for varicose ulcer. The numerous medicaments offered is evidence of the lack of knowledge regarding the basic problems involved. The rational treatment of ulcers should be based upon a sound understanding of the physiologic and mechanical factors responsible for movement of the blood through normal veins of the lower limbs, the abnormal factors present in the varicose state, the importance of other complicating conditions associated with varicose veins and ulcers and, above all, upon careful clinical observation and experience.

The author has employed over a period of years, practically every known medicament in the treatment of various types of ulcer cruris. In some patients the results were poor for two reasons: (1) Lack of appreciation of the basic problems involved, such as venous insufficiency, stasis and edema and (2) lack of specificity of the medicaments or ointments employed. Of great importance also in some cases is the presence of unrecognized epidermophytosis which is often present either as an etiologic factor or as a complicating factor.

PATHOGENESIS OF VARICOSE ULCER

Varicose ulcer, as the name implies, is associated with and due to varicose veins. In most instances the ulcer is located in the region of the medial malleolus, the most distal point of the great saphenous system. It is in this region that the hydrostatic pressure and stagnation of fluid in the tissues is most marked when the great saphenous vein is varicosed. Furthermore, this stagnant blood is oxygen-poor and metabolite-rich when compared with blood removed from normal veins in the lower limb. In addition, the exposed ankle is prone to minor traumas which frequently initiate the ulcer. Finally, it must be borne in mind that superficial thrombophlebitis is of common occurrence in patients with long standing varicose veins and the additional local phlebitis and lymphedema thus imposed upon an area of chronic stasis frequently act as the exciting factors in the production of ulcer.

The chronicity and intractability of some varicose ulcers is due to a number of changes in the tissues which may be briefly summarized: Once an ulcer starts, it always becomes infected. As a result, cellular infiltration takes place in the region of the ulcer margins and in the base or floor of the ulcer. Subsequently, fibrosis and cicatrical contraction of the tissues occur and the local capillary and arteriolar blood supply essential to growth of tissue and healing are shut off from the ulcer area. In addition, the usual pathologic changes, fibrosis and narrowing of the smaller surrounding blood and lymph vessels ensue with further embarrassment to the local tissue nutrition. Any type of treatment to be successful must be based upon a thorough knowledge of the underlying physiopathologic changes.

Some workers are opposed to injection therapy of varicose veins in the presence of ulcers and they usually defer such treatment until the ulcer is clean or has healed completely. When large varicose veins with demonstrable reverse flow are present, it has been the author's practice to operate (high ligation and division of the

great saphenous vein) early rather than late, regardless of whatever other local therapy is carried out. This operation frequently shortens the period of pain and disability to a great extent.

When dermatitis and enzema due to congestion of the skin in the presence of varices are present, bed rest for a short period of time, elevation and the application of Domeboro wet dressings 1:20 usually clear up the condition quickly. Domeboro tablets offer a convenient and excellent method of preparing Burow's solution. One crushed tablet dissolved in 1 pint of water makes a stable, buffered and non-irritating Burow's solution of 1:20 strength. Generally, the use of oiled silk or any other impervious material over the wet dressing should be avoided. Nothing in medicine has equalled the effectiveness of the Domeboro wet dressings in these patients with dermatitis and eczema. The comfort obtained almost immediately by their routine use is sometimes astonishing.

When considerable infection or epidermophytosis are present as complicating factors, wet dressings of potassium permanganate 1:5000 applied for a few days at the beginning of treatment may prove effective in preparing the area for subsequent Daxalan and Dome boot therapy.*

CHEMISTRY AND PHARMACOLOGY OF CRUDE
COAL TAR.

Coal tar (Pix lithanthracis) is a chemical storehouse of medicinal treasures. Investigation has proven that coal tar exhibits astringent, antiseptic and antiparasitic properties. In addition, it also exerts antipruritic, vasoconstrictive and keratoplastic effects as well as a definitely beneficial action on underlying cellular changes in various skin diseases. Many important therapeutic discoveries of recent decades have resulted from the numerous investigations in the chemistry and pharmacology of this black-appearing substance. It is

therefore one of the most valuable remedies employed in dermatology.

Brocq,[2] a European dermatologist, first advocated the use of crude coal tar in the treatment of mosit varicose ulcers. White[1] is credited with the introduction of the use of coal tar in this country and his published studies are most illuminating. As crude coal tar became more popular, it was pointed out that coal tar varied greatly in composition and consequently in pharmacologic action. These physical and chemical differences of coal tars derived from various sources were emphasized by different investigators.

Research has proven that for the manufacture of a suitable coal tar product, the source of crude coal tar should always be the same. It should contain a minimum of naphthalene and other objectionable substances and a sufficient amount of anthracene oil, heavy oil and pitch to exert keratoplastic, keratolytic, antiseptic, astringent and antipruritic action. The antiseptic and antipruritic effects depend chiefly on naphthalene, phenols and cresols. The keratoplastic effect is dependent on the presence of methylnaphthalene, dinaphthalene, xylenol and naphthol. The reducing action is closely related to its keratoplastic effect. The ability of coal tar to penetrate the epidermis depends on the presence of distillation products with high boiling points, especially methylnaphthalene. It abstracts oxygen from the skin, thereby inhibiting mitosis and resulting in a numerical and dimensional decrease in the cells of the rete malpighian and corneous layers.

The introduction of synthetic tars, distillates, so-called distillates, filtrates, and the solution of coal tar in fatty and nonfatty bases as a substitute for the whole crude coal tar product, have not been satisfactory. Therefore, the search for a satisfactory crude coal tar product to use in our clinic was instituted. Our attention was soon focused on a rigidly standardized, uniformly colored, aged crude coal tar product, called Daxalan,* used by dermatologists.

* The Daxalan ointment and the Dome boots employed in this study were furnished through the courtesy of Dome Chemicals, Inc., New York, N. Y.

Daxalan was first reported in the literature by Combes[3] in 1943, and again in 1944.[4] These observations have since been confirmed by other eminent authorities. With this background in mind, Daxalan was first employed clinically for all types of ulcer cruris in the Department of Peripheral Vascular Diseases at the New York Polyclinic Hospital.

Daxalan has the following formula:

Crude coal tar	2.88
Zinc oxide	5.37
Starch	53.75
Petrolatum, Q. S. ad	100.00

It is made by a special process and is aged for six months. This results in a smooth, homogenous ointment, uniformly dark in color, with no trace of coal tar flecks or small, black particles.

It is advisable to know that occasional reactions may be encountered when employing daxalan. Combes[3] has described the reactions and the precautions to be observed which are worthy of repetition here: (1) Avoid exposure to direct sunlight of the area to which tar is applied or has recently been applied. Tar is a photosensitizer, owing to the presence of acridine and the anthracene oils; (2) avoid prolonged use of tar on hairy areas. Folliculitis may occur, owing to pitch and paraffin oils; (3) avoid changing the dressing often; (4) do not apply tar to infected or impetiginized lesions; (5) do not apply tar to more than one-quarter of the body surface at one time. There is danger of absorption, since coal tars contain from 2 per cent to 4 per cent phenol; (6) be on the lookout for sensitivity after prolonged use; and (7) keep coal tar and coal tar pastes in carefully sealed containers to avoid evaporation of volatile ingredients which constitute a valuable part of their efficiency.

As soon as the skin clears and edema is reduced, ambulatory treatment is instituted. In many cases, the treatment is started without much preliminary cleansing or treatment other than the removal of the scaling skin by means of mineral oil or Domolene ointment (vaseline with a wetting agent). No benzene or ether is employed for cleansing since they are irritating and dry the area too greatly.

TREATMENT BY DAXALAN OINTMENT AND DOME PASTE BOOT

The objectives of this treatment are (1) to combat local infection and to stimulate healing; (2) to overcome the venous insufficiency stasis and edema present in most of the patients encountered in practice.

The first objective is attained by the daxalan ointment and the second by the principle of compression so effectively accomplished by the application of the Dome boot, a modified Unna's paste boot.

When the skin has been cleansed, a thick application of daxalan ointment is made directly to the ulcer and the surrounding skin. Unscented talcum powder is then sprinkled on the surface of the ointment application and a smooth, evenly applied Dome boot is then placed on the limb from the toes to the knee in the following fashion: A circular turn is made around the foot and carried up to the ankle and heel, where several figure eight turns are necessary to cover the heel. At no time is the bandage given any reverse turns because, when the boot stiffens, this may press against the skin as a ridge and cause much discomfort. The bandage is cut after one and one-half turns as it ascends the leg, each turn covering one-half of the preceding turn. The bandage or boot is thus completed an inch or two below the knee. The bandage, if properly applied, should cover the average leg approximately three times. The bandage (boot) is allowed to harden or dry in the air and the patient is instructed to return in one week. If the atmosphere is humid, the boot may be covered with a roller gauze bandage after a short while to protect the patient's clothing. (Figs. 1 to 5.)

The patient is usually ambulant after the boot application and is encouraged to remain so as far as possible. After one week, the boot is cut, the ulcer and surrounding skin cleansed with mineral oil or Domolene and a second application of daxalan ointment, talcum powder and Dome boot is

made. If the patient is comfortable, the second boot may be worn for a period of two weeks. The same routine is repeated until the ulcer is firmly healed. Occasionally, a yellowish or brownish stain will appear on the surface of the boot in the region of the ulcer. This may be covered with a little gauze which can be changed whenever desired.

During the course of treatment described above it will be noticed that when the boots are being changed the new skin appears
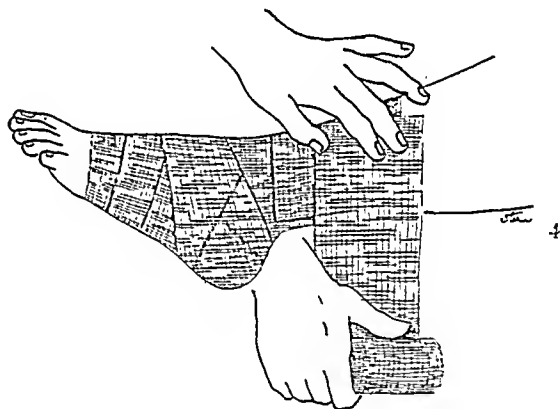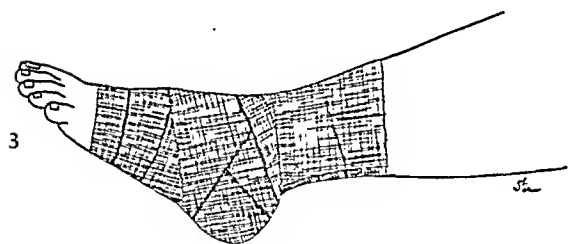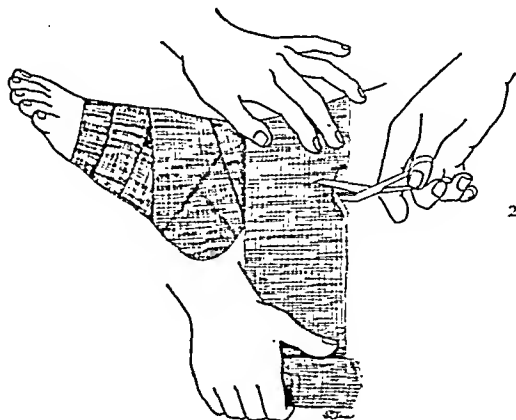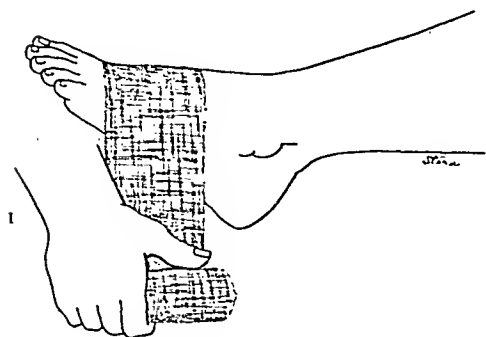
FIG. 1. The Dome Boot (bandage) is started with one horizontal turn applied around the foot and, when completed, the bandage is directed obliquely over the heel, etc.
FIG. 2. After the heel has been adequately covered the same method of application, i. e., one horizontal turn of the bandage followed by an oblique half turn and cut is carried out. This insures a flat application of the bandage to the skin surface.
FIG. 3. The bandage after it has been cut as described under Figure 2.
FIG. 4. The bandage is continued as described in text with a horizontal turn encircling the limb and a one-half oblique turn, after which the bandage is again cut and the same technic is carried out until the calf is covered.
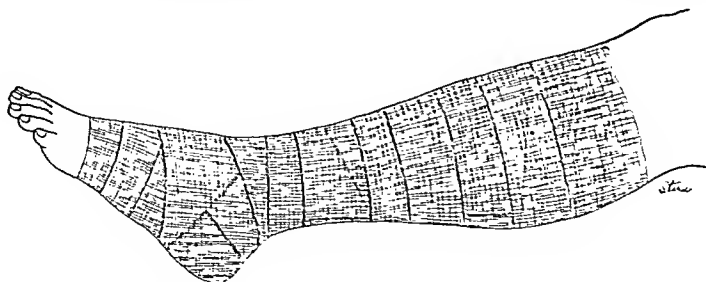
FIG. 5. This illustrates the completed bandage which always must cover the leg to a point well above the calf, otherwise the bandage may slip toward the ankle.
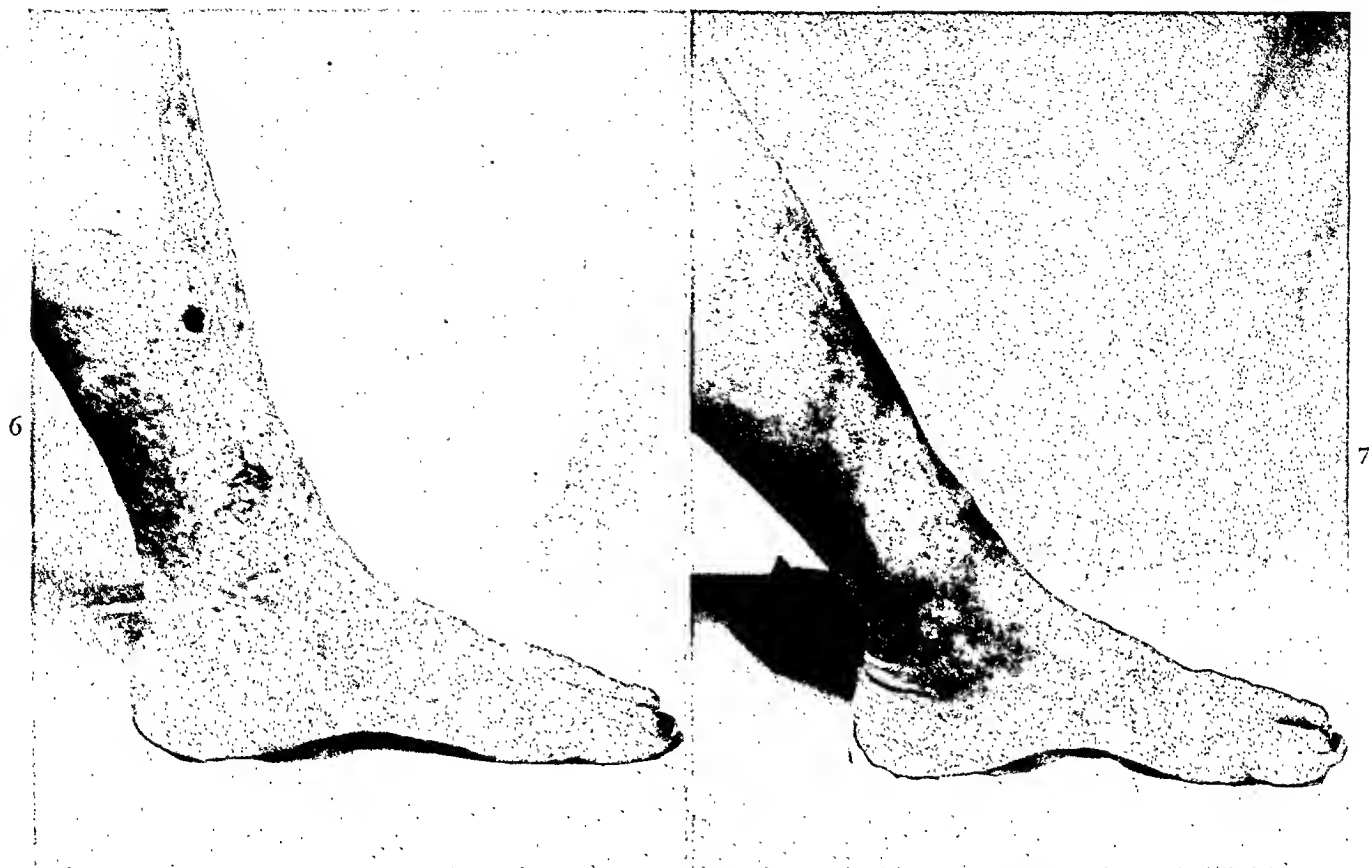
Fig. 6. Case No. 588-46, January 31, 1946. F. L., aged thirty-six, a married female with six children, was admitted to the New York Polyclinic Hospital out patient department on January 22, 1946 with a history of chronic, recurring ulcers of the left leg and an episode of post partum thrombophlebitis antedating the appearance of the ulcers. The history was otherwise irrelevant with the exception of a probable background of faulty nutrition since the patient was extremely poor and was obliged to do domestic work to supplement the family income. Examination disclosed multiple ulcers of the medial aspect of the left leg with considerable dermatitis and eczema. Edema 2 plus. The blood Wassermann test was negative. Treatment for the first fleck consisted of bed rest, elevation and Domeboro (1:20) wet dressings. On January 31, 1946 Daxalian ointment and the Dome boot were applied in the manner already described. The patient returned in one week, the boot was removed, the skin was cleansed with Domolene and a second treatment of the same type was given. In addition, Protinex (defatted, wheat germ embryo containing 37 per cent amino acids and vitamin B complex) and ascorbic acid three times a day after eating were prescribed.

Fig. 7. Same case as in Figure 6. February 14, 1946. Two weeks after commencing treatment there was complete healing of the ulcers noted in Figure 1. Only two applications of the Daxalan ointment and the Dome boot had been made in this case. Note the firm, white skin covering the previously ulcerated areas.

very firm and white. This should not be disturbed by too vigorous cleansing each time, but rather by gentle massage with the Domolene or mineral oil, in order to remove the old ointment before making a new application. After the ulcer has healed, particularly in the case of larger ulcer with edema, the treatment may be continued for several weeks to allow the new skin to become very firm.

At this time also, the surgeon will take cognizance of the underlying conditions responsible for the ulcer and direct his treatment with the idea of eliminating, as far as possible, the fundamental etiologic factors. During treatment with daxalan ointment and the Dome boot these conditions are adequately controlled; the daxalan ointment by stimulating rapid epithelization and overcoming infection and the Dome boot by correcting the underlying stasis and edema by the principle of compression. It is advisable to point out that the optimum time to apply the treatment is during the morning hours before the patient's limb has had a chance to become edematous.

Figures 6 to 15 illustrate several types of leg ulcers, all of which were treated by the method described. The results obtained in

FIG. 8. Same case as in Figure 6. March 14, 1947. Follow-up photograph four weeks after healing and completion of treatment; six weeks after commencing treatment. Note the firm-textured skin.

FIG. 9. Case No. 7322-44. February 7, 1946. M. D., aged forty-three, a colored female, married with two children, was admitted to the New York Polyclinic Hospital out patient department on September 21, 1944. The only noteworthy incident in this patient's past history was a spontaneous cerebral hemorrhage with resultant right hemiplegia at the age of thirty. She had been under treatment in the medical clinic for this condition as well as hypertension. The blood Wassermann was negative. Various ointment applications were made without any appreciable improvement. Two weeks prior to the time this photograph was taken Domeboro wet dressings (1:20) were applied and some healing is noted around the periphery of the ulcer. In addition the granulations appeared much cleaner. On February 7, 1946, the date of this photograph, the Daxalan ointment-Dome boot treatment was started and the dressings were changed weekly. Protinex and ascorbic acid were given three times a day after eating.
FIG. 10. Same case as in Figure 9. March 21, 1946. Six weeks after beginning the Daxalan ointment-Dome boot treatment there was complete, firm healing of the ulcer of the lateral aspect of the left leg.

Fig. 11. Case No. 10965-40. January 31, 1946. Medial surface of the left leg at commencement of treatment. This figure and Figures 12, 13, 14 and 15 illustrate, in an elderly female, the rather remarkable progress and healing of multiple ulcers of the right leg of more than fifteen years' duration. S. M., seventy-five years of age and a self-supporting janitress, had been a patient in various departments of the New York Polyclinic Hospital. She was admitted to the vascular clinic on August 29, 1940 with a history of chronic, recurring ulcers of the right leg for the past fifteen years. Various types of medicaments were applied, including most of the ointments advocated for the treatment of these conditions, but with no appreciable improvement.

On January 31, 1946 the Daxalan ointment-Dome boot treatment was started and the ointment and boot changed once weekly thereafter. This was supplemented with Protinex and ascorbic acid three times daily. Healing of the ulcers of the lateral surface was noted on March 14, 1946, and of the large ulcer on the medial surface, April 11, 1946.

Fig. 12. Same case as in Figure 11. January 31, 1946. Lateral surface of the right leg at onset of treatment.

FIG. 13. Same case as in Figure 11. March 14, 1946. Medial surface of the right leg six weeks after starting treatment showing considerable epithelization from the ulcer margins toward the center of the ulcer. Note the healthy, red granulations in the unhealed central portion.

FIG. 14. Same case as in Figure 11. March 14, 1946, six weeks after the treatment was instituted, showing complete healing of the ulcers of the lateral surface of the right leg.

each patient as well as complementary treatment are described in the text accompanying the photographs.

SUMMARY AND CONCLUSIONS

One-hundred forty-seven subjects with chronic leg ulcers of varying etiologic backgrounds were treated by the daxalan-Dome boot technic over a period of eighteen months. Most of the patients were of the type usually described as "varicose," but a large proportion of the patients presented edema due to thrombophlebitis or chronic venous insufficiency associated with extensive varicose veins of long standing.

The maximum period of time required to effect a cure of any individual in this series was ten weeks and the minimum, two weeks.

Dermatitis if present, was always treated by simple, nonirritating, local therapy consisting of Domeboro ($\frac{1}{20}$) wet dressings. These proved vastly superior to any other
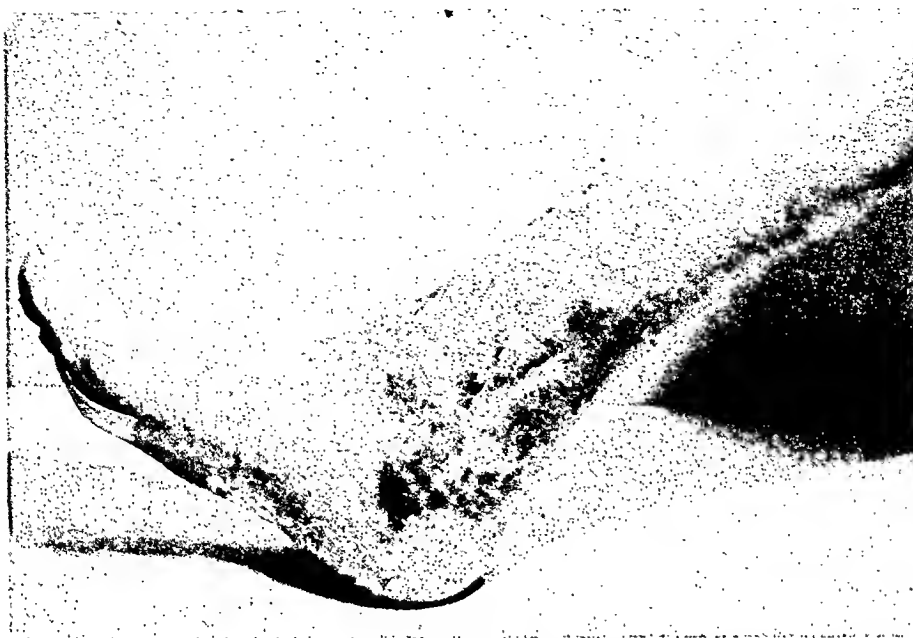
FIG. 15. Same case as Figure 11. April 11, 1946, ten weeks after commencing treatment with Daxalan ointment and Dome boot, showing complete healing of the large ulcer of the medial surface of the right leg. Treatment throughout was on an ambulatory basis and this elderly patient was permitted to do her usual work as a janitress.

type of treatment. Subsequent treatment with daxalan and the Dome boot sufficed to cure the ulcer.

Sensitivity or allergy to daxalan was encountered in only two patients in this series and in both instances treatment with daxalan ointment was immediately discontinued. In this connection the author wishes to point out the importance of performing a patch test on each new patient, care being taken to do this test in an area of diseased skin. If no irritation results from the medication, one may employ daxalan with safety. Furthermore, if one so desires, the half-strength daxalan, known as daxalan-pediatric, may be employed at first and if no irritation is observed, later treatments may be carried out with regular daxalan. The precautions mentioned by Combes should be observed whenever daxalan is employed for a long period of time.

While other satisfactory methods of treatment for the cure of leg ulcers have been utilized by the author, the daxalan-Dome boot method has proven wholly satisfactory as an ambulatory method of treatment for these distressing conditions. It permits many disabled and partially disabled individuals to engage in their usual work without discomfort.

The daxalan-Dome boot method of treatment conforms to sound physiologic principles; the ointment by its antiseptic, stimulating and epithelizing qualities and the boot, because of its semi-rigid character, by compressing the limb, eliminating edema and causing drainage from the ulcerated area to diminish. It seems almost needless to repeat that healing of a leg ulcer in an edematous limb is almost impossible unless the "water-logged" condition is eliminated. A smooth, properly applied boot accomplishes this nicely.

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TREATMENT OF CARCINOMA OF THE BREAST*

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THE present study of 324 patients with carcinoma of the breast was made to determine (1) the clinical error of axillary node involvement proven histologically, (2) the effect of duration of symptoms on survival rate, (3) the relative merits of

Paget's disease, mucoid and comedo carcinomas were relatively rare. This classification is useful, although probably not as accurate as one which stresses the origin of the tumor.[1,10]

The term five-year survival is used in-

TABLE I

CLINICAL ERROR IN DIAGNOSIS OF AXILLARY NODES IN GROUPS I AND II

	No.		No.		
Clinically negative nodes ...	85	Pathologically positive nodes..	27	Per cent of error..........	31.8
Clinically positive nodes. ...	136	Pathologically negative nodes..	36	....	26.4
				Average error both groups...	28.5

various types of treatment, (4) the comparison of the survival rate after radical and subradical surgery, (5) the survival rate according to the histological type of carcinoma, (6) the incidence and the effect on survivals of the site of the primary involvement, (7) the percentage of local recurrence in each clinical group and (8) the fate of patients who survived for five and ten years.

A few definitions are necessary to further outline the discussion. Patients have been classified in four main groups as follows: Group I, Carcinoma confined to the breast; Group II, metastases to the axillary nodes only; Group III, metastases beyond the axillary nodes; Group IV, hopeless cases.

The group designation was made only on histological proof from removed tissue, an important point which has been emphasized previously.[4,5,15] Three main pathological groups were used in this classification: (1) Scirrhous, fibrous stroma predominating; (2) medullary, epithelial elements predominating; (3) adeno or ductal, intraductal and intracystic origin or carcinoma with definite adenomatous structure.

stead of "cure" since many who lived five years had carcinoma present at the end of the five-year period and many later developed recurrences and died after five years.

Of the 324 cases studied, 24 or 7.4 per cent were not traceable up to the end of five years and 5 more were lost later, making a follow-up percentage of 91 per cent. Two hundred fifty-seven operations were performed by ten different surgeons. Seventy-five per cent of these operations were performed by Dr. T. G. Orr. There were ten incidental deaths (deaths due to other diseases) and five postoperative deaths. The operative mortality was 2.4 per cent. These fifteen deaths were excluded from most of the analyses.

A definite clinical error in the estimation of axillary node involvement is at once apparent from a study of Table I. Twenty-seven of eighty-five patients said to have non-palpable nodes had definite carcinoma in the nodes, making an error of 31.8 per cent. In 136 patients with nodes palpably enlarged and thought to have carcinoma, thirty-six were found not to have axillary involvement on section, an error of 26.4

per cent. Clinical findings for determining the presence or absence of carcinoma in nodes were wrong in about one out of three patients. Other authors[11,14] have reported 31 per cent and 28 per cent of errors in the clinical appraisal of axillary nodes.

TABLE II

COMPARISON IN GROUPS I AND II OF DURATION OF SYMPTOMS IN MONTHS BEFORE ADMISSION TO AVERAGE AGE ON ADMISSION OF FIVE-YEAR SURVIVALS AND THE DEATHS

	Duration before Admission in 5-year Survivals, Mo.	Duration before Admission in Death Cases, Mo.	Average Age of 5-year Survivals on Admission, Yr.	Average Age of Death Cases on Admission, Yr.
Group I...	11.3	12.1	52.4	55.1
Group II...	9.9	12.3	52.7	50.3

Comparing five-year survivals in Groups I and II the duration of symptoms before admission showed no significant difference. (Table II.) The average ages of five-year survivals in Groups I and II were nearly identical but in the deaths the average age was five years younger in Group II than in Group I. This was as expected from the known fact that, as a rule, carcinoma in the younger age group metastasizes and kills more rapidly.

Radical surgery is certainly the only treatment which can be expected to effect many cures. Considering all groups together, a comparison of the five-year survival rate in patients with radical surgery alone, with radical surgery plus preoperative or postoperative irradiation, or both, shows nothing very significant. Radical mastectomy alone plus preoperative irradiation showed almost identical survival rates. (Table III.)

With postoperative irradiation the survival rate was increased by 5 per cent. With radical surgery followed by postoperative irradiation most authors have shown the same[5] or greater increase,[6,11,12] although some[1,4] reported poorer results. The increase with postoperative irradiation of survivals occurred in Group II but not in Group I. Undoubtedly cases wherein this treatment was used were initially more far advanced. The twenty-seven simple amputations performed were almost entirely done for palliation, yet over 18 per cent of these survived five years. Daland[2] in reporting one hundred untreated cases found twenty-two alive after five years and five still living after ten years.

TABLE III

COMPARISON OF FIVE-YEAR SURVIVALS ACCORDING TO THE METHOD OF TREATMENT USED*

	University of Kansas Hospitals			Operation Elsewhere		
	Total No.	No. 5-year Survivals	Per Cent of 5-year Survivals	Total No.	No. 5-year Survivals	Per Cent of 5-year Survivals
Radical mastectomy alone.............	48	21	43.7	11	7	63.7
Radical + preop. irradiation....................	39	17	43.6	1	0	0
Radical + postop. irradiation....................	72	35	48.6	13	5	38.5
Radical + pre and postop. irradiation...........	44	15	32.6	1	0	0
Simple mastectomy alone.....................	9	2	22.2	6	1	16.7
Simple + preop. irradiation...................	5	2	40			
Simple + postop. irradiation..................	5	0	0	4	0	0
Simple + pre and postop. irradiation...........	8	1	12.5	1	0	0
Total.........................	230	93	44.3			

* No incidental deaths, operative deaths or cases of doubt included.

In some patients the routine radical operation was modified by leaving intact one or both pectoral muscles. The effect on survival rate of this subradical amputation, consisting of completely removing all breast tissue and cleaning out the axillary

radical operation, mainly for palliation, lived five years.

A study of the influence of the various histological types of carcinoma on the survival rate was made. Comparing average ages for the different histological types

TABLE IV

COMPARISON OF FIVE-YEAR SURVIVALS BY GROUPS I AND II ACCORDING TO RADICAL OR SUBRADICAL OPERATION*

	Group I			Group II		
	No. Operations	5-year Survivals	Per Cent of 5-year Survivals	No. Operations	5-year Survivals	Per Cent of 5-year Survivals
Radical.......	63	44	69.9	74	18	24.3
Subradical....	20	17	85	16	4	25
Total.......	83	61	73.5	90	22	24.4

* No case of operative deaths or incidental deaths are included.

TABLE V

COMPARISON OF HISTOLOGICAL TYPES BETWEEN INCIDENCE, AGE AND PERCENTAGE OF FIVE-YEAR SURVIVALS OF PATIENTS IN GROUPS I, II AND III*

Histologic Types	No.	Percentage Incidence	Average Age on Admission, Yrs.	Number of 5-year Survivals	Percentage of 5-year Survivals
Scirrhous.............	166	66.4	52.2	62	37.3
Medullary.............	16	6.4	50.7	6	37.5
Adeno (ductile).......	56	22.4	58.6	25	44.6
Mucoid and comedo...	6	2.4	57	4	66.7
Paget's disease........	4	1.6	54.8	2	50

* Only patients who had cases that supplied definite pathological diagnoses are included here. Five-year survivals quoted are those on which all types of surgery were performed. Incidental and operative deaths are not excluded.

contents, was studied by comparing the results with those after radical surgery in Groups I and II. (Table IV.) The selection of the subradical procedure was made for some patients who were aged or debilitated, who had a low grade malignancy such as malignant intracystic or intraductal papilloma with little invasion as proven by biopsy or who had concomitant disease of such severity as to modify the treatment. In Group I there were forty-four five-year survivals in sixty-three patients who had the radical operation, or 73 per cent, compared with seventeen five-year survivals in twenty patients who had the subradical amputation, or 85 per cent. In Group II eighteen of seventy-four patients, or 24.3 per cent, lived five years after radical operation as compared with four of sixteen, or 25 per cent, who underwent subradical surgery. It would seem that with proper patient selection the subradical operation is warranted and certainly produces less shock and a better functional result. Five-year survivals for Group I were 73.5 per cent, averaging both radical and subradical results as compared with 24.4 per cent for Group II. One patient in sixteen in Group III who submitted to

revealed nothing startling. Adeno, ductile, mucoid and comedo carcinoma occurred in the slightly older patients rather than scirrhous and medullary carcinoma. (Table V.) Of the three main types, scirrhous, medullary and adeno (ductile) carcinoma, the survival rate in the first two were practically identical but in adeno (ductile) carcinoma 7 per cent more patients lived five years. This was as expected. Mucoid and comedo carcinoma and Paget's disease were too few to be statistically significant but the relatively lower grade of their malignancy is known.

The primary site of the carcinoma in the breast reviewed to determine the incidence and the effect on the five-year survival rate with and without axillary involvement. (Table VI.) The upper outer quadrant was by far the most common primary site, occurring in 52.6 per cent of the total. Other primary sites were fairly equally distributed in the inner quadrants, the lower outer quadrant and beneath the nipple. It was interesting to find that the inner quadrants without axillary node involvement gave a fairly high percentage of

five-year survivals but a comparatively lower survival rate than the outer quadrants when the nodes contained cancer at the time of operation. Being further from the axilla, if nodal involvement is present in the inner quadrant group, metastases more

Most interesting was a follow-up study of the fate of the five and ten-year survivals. There were one hundred patients who had lived five years after treatment but ten were excluded since they died from causes other than carcinoma and six were not

TABLE VI

COMPARISON OF INCIDENCE AND FIVE-YEAR SURVIVALS ACCORDING TO LOCATION IN BREAST OF PATIENTS IN GROUPS I AND II*

Location	Total No.	Per Cent Incidence	No. in Group I	No. 5-year Survivals	Per Cent 5-year Survivals	No. in Group II	No. of 5-year Survivals	Per Cent 5-year Survivals
Upper outer quadrant	130	52.6	53	30	56.6	63	11	15.9
Lower outer quadrant	21	8.5	9	6	66.7	8	3	37.5
Upper inner quadrant	30	12.2	15	12	80	12	2	16.7
Lower inner quadrant	21	8.5	12	6	50	7	1	14.3
Beneath nipple	24	9.9	12	6	50	12	3	25
Total	226	....	101	60	59.4	102	20	19.6

* Only those patients are included in which the location was stated without question. All types of treatment are included.

likely have already taken place in vital organs. The importance of quadrant involvement has been noted previously.[7,9]

The problem of local recurrence in its relation to the type of surgery employed has been presented in studies from many clinics. Radical amputation with plastic closure (Handley) is employed almost routinely in this clinic. Only a very few mastectomies in this series required skin grafting. The local recurrence rate in eighty-three patients in Group I was 13.3 per cent as compared with 20 per cent of ninety patients in Group II and 44 per cent of sixteen patients in Group III. The over-all local recurrence rate including the Group III patients was 20 per cent. (Table VII.)

Excluding Group III the local recurrence rate was 16.0 per cent averaging the results in Groups I and II. These results are more favorable than percentages reported from large clinics[4,8] solely employing the Halsted type operation which is said to lessen the number of local recurrences and are about the average, 19.4 per cent, of several authors[4,13,14,15,17] employing plastic closure.

TABLE VII

INCIDENCE OF LOCAL RECURRENCE AFTER RADICAL OPERATION BY ALL GROUPS*

Group	No. of Operations	No. with Local Recurrence	Per Cent of Local Recurrence
I	83	11	13.3
II	90	18	20
III	16	7	44
All groups	189	36	20

* Deaths due to operation, deaths not due to carcinoma and patients with unknown pathology of axillary nodes are not included.

included as they had had their surgery performed elsewhere. Of the eighty-four remaining, twenty-five, or 29.8 per cent, have subsequently died from their original carcinoma and many of the eighty-four in the follow-up series are only one to three years past the five-year mark. Out of sixty-one patients in Group I five-year survivals 13, or 21.3 per cent, subsequently died of metastases. In Group II, 11 of 22 patients, or 50 per cent of the five-year survivals later died of the original carcinoma. Among twenty-two who had survived ten years,

nine have either succumbed or are living with their cancer at present. Two of the nine patients had a recurrence before the five-year mark, two between the five and ten-year periods and five developed metastases attributable to their original carcinoma after ten years apparent freedom from disease. Of Simmons[14] five-year survivals, 19 percent later developed recurrence.

SUMMARY

1. The clinical estimation of axillary involvement in carcinoma of the breast is in error in approximately one out of three patients.

2. The average duration of symptoms before admission is about the same in those patients with and without axillary metastases.

3. Radical surgery is the main therapeutic weapon to combat carcinoma of the breast. Postoperative irradiation will give a few more five-year survivals in Group II patients.

4. In properly selected patients, considering histology, age, condition and extent of the carcinoma, leaving one or both of the pectoral muscles does not lower the survival rate.

5. Scirrhous and medullary carcinoma are the most malignant, adeno is slightly less malignant and Paget's disease, mucoid and comedo carcinoma are the least malignant.

6. Carcinoma developing in inner quadrants of the breast has a better prognosis than those in the outer quadrants, unless the axillary nodes are involved in which case the reverse is true.

7. With radical surgery and plastic closure the local recurrence rate was 13.3 per cent in Group I, 20 per cent in Group II and 44 per cent in Group III. The average recurrence rate in Groups I and II was 16.0 per cent.

8. Nearly 30 per cent of the five-year survivals later died of carcinoma and nine of twenty-two ten-year survivals had at ten years or later showed a recurrence.

9. It is fully recognized that the study of such a small number of patients is not of great statistical value but presents the trend only in one clinic.

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TRAUMA TO THE REGION OF THE BURSA ANSERINA

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FRACTURE of a single bone of the leg without any associated subluxation at the upper or lower fibulotibial articulation was the usual injury sustained by soldiers of horse cavalry or mule pack outfits. These fractures caused by kicks of the animals were usually of the comminuted stellate type and showed on radiographic examination very little displacement of the main fragments. (Fig. 1.) Healing of these fractures occurred without any complications and did not result in impairment of motion at the ipsilateral knee or ankle.

Direct impact of the animal's hoof may also cause, but less frequently, a thickening of the soft tissues in the region of the bursa anserina* without fracturing the underlying bone. Such swellings may be long lasting and may simulate intra- or extra-articular lesions especially that of cysts of the semilunar cartilages. We had an opportunity to study three soldiers with thickening of the soft tissues in the region of the bursa anserina.[1-3] In one of the three, surgical intervention was necessary in order to effect a cure.

In this investigation, the clinical, radiographic and histological findings and differential diagnoses are presented primarily to discuss the unusual condition of chronic thickening of the soft tissues in the region of the bursa anserina as caused by direct trauma to the local part.

HISTORY AND PHYSICAL FINDINGS

Each of the three soldiers in question stated that they were struck by the hoof of an animal while participating in military

FIG. 1. Radiograph shows comminuted fracture of the upper third of the tibia. This was caused by a kick of a mule.

training. The kick caused a swelling on the medial aspect of the upper portion of the leg of each of the three soldiers. The three patients sought admission to the hospital from nine, thirty and forty-four days, respectively, after the injury because of persistent local pain and swelling. In the case of the patient who was examined nine days after the injury, we found a definite prominence along the inner aspect of the proximal portion of the right leg in the region of the bursa anserina. When the affected leg was fully flexed against active resistance the patient experienced severe pain over this swelling. There were five

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* The bursa anserina is situated superficially to the tibial collateral ligament on the upper medial surface of the tibia. The bursa is enclosed by the tendons of the sartorius, gracilis and semitendinosus.

FIG. 2. Radiographs (retouched) made in the anteroposterior and oblique projections show thickened soft tissues in the region of the bursa anserina (arrow). The three views are of the same knee.

degrees of restriction to active and passive extension of the affected leg. Tenderness was present along the course of the internal lateral ligament, as well as in the regions of the anterior and posterior horns of the internal semilunar cartilage.

As for the examination of the patient who was seen thirty days after the injury, a flat mass measuring the diameter of a silver dollar was noted on the upper medial aspect of the leg; it extended to the level of the upper articular surface of the tibia. This mass was firm, round, tender to the touch and freely moveable. Active flexion of the leg against resistance caused pain over the described swelling. The range of active and passive flexion and extension of the leg was within normal limits. As for the examination of the patient seen forty-four days after the injury we also observed a mass, the size of a small lemon, situated on the medial aspect of the upper portion of the leg and knee. This swelling was tender to the touch and did not interfere with the range of active and passive movements of the left leg. There was no effusion in the

knee joint. Active flexion of the leg against resistance, however, produced local discomfort over the swelling. No instability was noted of the bones comprising the knee joints of this or of the other two patients in question. Similarly, there was no evidence of enlargement of any other bursae about the knee of these three patients.

Radiographic examinations were made of the knee and leg in the anteroposterior, lateral and oblique projections with the bone and soft tissue technics. These studies demonstrated thickening of the soft tissues on the medial aspect of the upper portion of the tibia and knee. At no time was there any evidence of periostitis of the contiguous tibia or ossification or calcification of the tendons in or about the bursa anserina or the lateral ligaments of the knee joint. (Fig. 2.)

The patients were placed in bed and warm or cold compresses were applied to the affected enlarged, tender areas. In one patient where there was an inability to actively completely extend the leg traction to the lower extremity was instituted. In addition, active non-weight bearing quadriceps exercises were initiated soon after admission to the hospital. These three patients were hospitalized for periods varying from thirty to sixty days, and except for one in whom surgical intervention was necessary, resolution of the swelling was a slow process. When the two patients who received conservative therapy were permitted to do excessive amounts of walking or marching, the swelling in the region of the bursa anserina reappeared. However, complete bed rest plus non-weight bearing exercises to the quadriceps led to an almost complete resolution of the prominence. Physical therapy, such as whirlpool baths and infrared radiation, were used effectively to lessen the size of the swelling of the affected region.

In the instance of the patient seen forty-four days after the injury who presented a swelling the size of a small lemon, physiotherapy and periods of rest for thirty days had little effect on the size of the mass. For
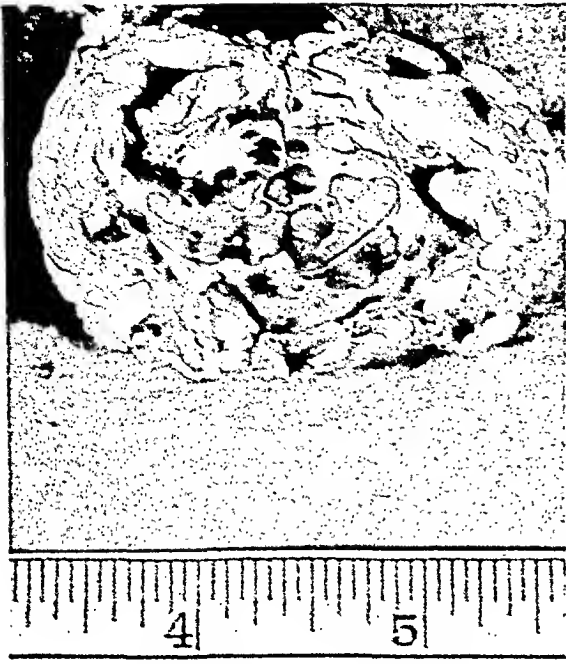
FIG. 3. Photograph of one wall of the subcutaneous adventitious bursa reveals the presence of currant jelly-colored elevations in a mosaic arrangement. The polyp is attached to another wall and is not seen in this photograph.

this reason surgical intervention was followed. The exploration revealed that the mass consisted of a subcutaneous adventitious bursa which had a direct channel with the cavity of the contiguous bursa anserina. The subcutaneous bursa was walled by scarred subcutaneous fat and its cavity held about 25 cc. of clear fluid. At the upper pole of this bursal lining was a pendant polyp. The lining of the bursal wall which presented raised circumscribed areas of a currant jelly color joined with the wall of the bursa anserina. In the latter, about 15 cc. of clear fluid and numerous pendant and detached villi were found. The subcutaneous bursa and its polyp as well as the lining of the bursa anserina were removed *in toto*. Resistance quadriceps exercises were initiated forty-eight hours after the operation.

Pathological Findings. The specimen as received measured when opened 4 by 4 by ⅟₁₆ cm. and the polyp measured 2½ by 1½ by ½ cm. The lining of the wall of the subcutaneous bursa consisted of brownish-red elevations almost resembling the appearance of a mosaic arrangement of fungus

colonies. Some of these single colored areas measured from 0.2 to 0.5 cm. in diameter. (Fig. 3.) Sections of the polyp showed that it consisted primarily of fatty tissue. The lining of the bursa anserina was three times thicker than normal and on its synovial surface were numerous small heart-shaped villi. The microscopic examination showed that the colored areas in the wall of the subcutaneous bursa were the result of deposition of blood pigment and necrosis of part of the subcutaneous fat. The wall of the bursa anserina was thickened and on its lining were numerous small villi. No evidence of infection was present.

The program of conservative treatment and restriction of activities as pursued in the other two patients caused a diminution in the size of the swelling about the bursa anserina. When these patients attempted to perform their regular field duties there was a recurrence of the enlargement of the affected bursa. For this reason they were reassigned to duties which did not require excessive use of their knees. When this was accomplished they had no further difficulties with their legs. In regard to the third patient in whom operative intervention was performed a follow-up examination sixty days after the operation showed that the power of the extensor apparatus of the leg was equal to that of the normal side. No recurrence of swelling was noted in the region of the scar after rigorous exercises. This patient was, therefore, returned to general duty.

COMMENTS

This study has revealed that trauma to the upper portion of the leg may cause hemorrhage in and necrosis of the subcutaneous tissues about the bursa anserina. It is known that the subcutaneous tissues in the upper part of the leg contain a considerable amount of fat which permits free movement of the skin. The looseness of the subcutaneous tissue over the tibia also allows for the inspissation of large amounts

of blood which may lead to the formation of adventitious bursae. When this occurs, as was demonstrated in the one patient in whom surgical intervention was followed, the adventitious subcutaneous bursa may join with that of the contiguous bursa anserina. These abnormal prominences about the medial aspect of the upper leg may lead to an erroneous diagnosis of extra-articular cysts in the semilunar cartilage, bursa of the tibial collateral ligament, osteochondroma of the tibia or ganglions about the hamstring tendons, unless one keeps in mind the possibility of adventitious bursa in conjunction with the bursa anserina. Radiographic examinations made in the oblique view of these three patients disclosed a distinct increase in the thickness of the subcutaneous tissues directly over the medial surface of the proximal end of the tibia and the absence of any distinct localized soft tissue shadow at the level of the semilunar cartilages.

When the soft tissue mass in question diminished in size on rest and physiotherapy, change in the duty status to one which does not entail excessive use of the limbs is indicated. However, when the mass does not alter in size subsequent to the limitation of activities and physiotherapy for at least a thirty-day period, one must suspect villonodular synovitis of the bursal lining.

Surgical intervention is necessary to effect a cure in this instance.

CONCLUSION

Severe trauma to the upper portion of the leg as caused by the kick of an animal may result in a fracture of a single bone or a thickening of the soft tissues about the bursa anserina. The latter may be mistaken for an extra-articular cyst or ganglion. When the soft tissues are involved as was noted in three patients the underlying tibia apparently showed no evidence of periostitis. The swelling of the soft tissues may be the result of an adventitious subcutaneous bursa secondary to necrosis of the underlying fat and inspissation of blood. This adventitious bursa may be directly linked to the cavity of the bursa anserina. When the soft tissue prominence does not recede with rest or limitation of activity, as was noted in one patient, surgical removal of the mass is indicated. Conservative therapy, such as physiotherapy, traction and limitation of activity led to a definite amelioration of the symptoms in the other two patients.

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COMPARATIVE EVALUATION OF THE EFFECTS OF TALCUM AND A NEW ABSORBABLE SUBSTITUTE ON SURGICAL GLOVES*

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THE potential hazards of talcum as a lubricant for surgical gloves have long been a matter of concern. The effects of talc within the peritoneal cavity were investigated in 1919 by Hertzler,[1] and subsequently by Haythorn[2] and Antopol.[3] In 1936 Owen[4] conducted an intensive investigation of the subject and suggested that talc be removed completely from the surface of the gloves before operating. Her findings found support in the work of Feinberg,[5] Ramsey,[6,7] Byron and Welch,[8] Weed and Groves[9] and German.[10]

Lichtman, McDonald, Dixon and Mann,[11] in the course of an extensive study of birefringence in tissues and an investigation of changes in tissues in human beings and laboratory animals produced by doubly refractile foreign bodies made visible by polarized light, offered incontrovertible evidence of the local irritant action of talcum. They found that the characteristic reaction in human tissues consisted of the formation of pseudotubercles and that crystals of talc implanted in artificially created fistulas in dogs caused a persistence of the fistulas. Their findings lent considerable emphasis to the need for an adequate substitute for this irritating substance.

Seelig[12,13] and Seelig, Verda and Kidd[14,15] who have repeatedly warned of the dangers of talc in operative wounds, offered potassium bitartrate and, subsequently, an experimentally produced formaldehyde-treated starch as possible substitutes. Both of these substances have been found to be unsatisfactory for a variety of reasons.

In view of the grave dangers attendant upon the use of talcum, a great deal of effort has been devoted to the search for a satisfactory substitute. Of many compounds tested by the authors and others, in the laboratory and clinically, one has proved so promising as to warrant its introduction for widespread clinical use under practical operating room conditions. This substance consists of a mixture of amylose and amylopectin, derived from corn starch, which has been treated by physical and chemical means to improve its lubricating value and to prevent gelatinization when autoclaved; a small amount (1 per cent) of magnesium oxide is included for the purpose of further improving the flow properties of the mixture. For the sake of convenience this preparation has been designated Biosorb absorbable powder.†

Lee and Lehman[16] found that this material possessed excellent physical qualities of flow and fineness which were largely unaffected by autoclaving and that it was completely absorbable from the peritoneum without inflammatory reaction and without the formation of adhesions. Their clinical experience with the powder, as well as that of Walkling and Lindenmuth,[17] proved eminently satisfactory. MacQuiddy and Tollman[18] who investigated the anaphylactogenic properties of biosorb powder were unable to find any sensitivity to this modified starch in any of the humans tested, nor was it possible to produce a state of sensitivity in animals when this

substance was used as the anaphylactogen. Their findings further supported the observations of others that this material is non-irritating, is absorbed readily from the peritoneal cavity and appears to be an entirely satisfactory replacement for talcum powder for surgical purposes.

In view of the potential importance of this new powder and the fact that some of the previously offered substitutes (notably potassium bitartrate) have proven inadequate, in part because of their deleterious effects on rubber gloves, it was deemed of interest to conduct a detailed comparison of the relative effects of talcum powder and Biosorb powder on the rates of deterioration of several brands of gloves prepared with these substances and subjected to repeated autoclavings.

It is, of course, well known that steam sterilization alone produces considerable deterioration of surgical rubber gloves, but the question of importance to be determined was whether the rate of this deterioration is accelerated when the gloves are treated with Biosorb instead of talc.

Surgical gloves were purchased from six manufacturers; they comprised several types, including those fabricated from neoprene, white latex and brown latex. The gloves were divided equally into two sets, those in one set being treated in all subsequent procedures with talcum powder and those in the other with Biosorb powder. The preparation of the gloves before each autoclaving followed standard hospital practice as closely as possible. The gloves were washed thoroughly with soap and water, air dried and dusted inside and out with the lubricant powder. A pledget of powder-impregnated gauze was then placed in the cuff of the right hand glove, each pair placed in linen glove folders and autoclaved at 15 pounds pressure (240°F.) for thirty minutes.

After each sterilization the gloves were examined for flaws during moderate distension, washed free of powder and allowed to air dry for a period of twenty-four hours; at the end of this period they again

were prepared, respectively, with talcum or Biosorb and autoclaved, the cycle being repeated the requisite number of times.

Tensile strength measurements were made on each set of gloves before sterilization and after groups of three, six and nine consecutive autoclavings. While a number of possible methods of determining the degree of deterioration suggested themselves, it was thought that measurement of the tensile strength probably was the most accurate quantitatively with the facilities available to us. This factor was correlated with the ability of the glove to withstand the stress and strain of application under clinical conditions as a qualitative measure of the point of failure under conditions of practicable use. It was found that, in general, when the tensile strength became less than 1.5 pounds the likelihood of tearing during the act of putting on the gloves, or during subsequent manipulations, was great.

The tensile strengths were determined on sections measuring 0.25 by 2.0 inches (.635 by 5.08 cm.) cut from the gloves with a special die. A Scott tensile strength tester was used with the jaws set at 0.5 inch (1.27 cm.) apart and the results were recorded as pounds pull at the moment of rupture. Since the material used in the manufacture of surgical gloves is not of uniform thickness, thickness measurements were made on each section tested, using a standard thickness micrometer of the type used in determining the diameter of catgut. These determinations were correlated with the tensile strength results, which thus are corrected for differences in thickness of the samples tested; 0.01 inch (0.25 mm.) was taken as the standard thickness. The results are summarized in Table I. Each result listed in the table represents the average of five determinations per glove. We wish to emphasize that we are not offering these data as absolute values indicating true tensile strengths. The principal interest is in determining relative effects; since each determination was obtained in an identical

TABLE I

COMPARATIVE TENSILE STRENGTH OF SURGEON'S RUBBER GLOVES WHEN TREATED WITH TALCUM AND BIOSORB ABSORBABLE POWDER — POUNDS PULL OF RUBBER SECTION 0.25 BY 0.01 INCH

Brand	I			II			III	IV	V	VI	
Type	White Latex	Brown Latex	Brown Latex	White Latex	Brown Latex	Neoprene	Brown Latex	Brown Latex	White Latex	White Latex	Grand Averages
Control (without any powder and not sterilized)	4.9	2.6	4.4	2.6	4.9	6.6	4.3	2.4	5.2	5.0	
	3.8	4.3	4.3	5.0	5.1	5.2	4.1	2.0	4.5	5.2	
	4.0	3.8	5.2	4.9	5.5	2.8	4.4	2.6	5.1	5.2	
	3.6	2.8	5.1	5.8	3.5	3.0	4.2	2.6	5.0	4.1	
	5.4	2.8	4.2	3.3	4.7	5.6	4.8	2.0	5.3	5.4	
$\bar{X}$	4.9	3.3	4.6	4.3	4.7	4.6	4.4	2.4	5.0	5.0	$\bar{\bar{X}}$ 4.32
Biosorb absorbable powder 3 sterilizations	4.8	1.0	2.0	4.3	4.7	4.6	4.2	2.0	4.0	5.0	
	3.1	2.1	3.1	3.3			4.4	2.6	2.5	1.4	
	2.9	2.2	3.1	3.4	3.6		4.8	2.6	3.5	1.4	
	4.0	2.4	4.0	4.0	4.0	1.7		2.0	5.3	1.2	
	3.2	2.0	3.9	3.7	4.5	2.1	3.8			1.5	
$\bar{X}$	3.6	2.1	3.4	3.7	5.5	5.1	3.6	1.8		1.2	$\bar{\bar{X}}$ 3.04
Talcum powder 3 sterilizations	3.6	2.2	3.4	3.6	4.2	2.2	2.2	2.0	3.0		
	3.2	2.0	3.7		4.4	8.2	3.6	1.6	4.0	1.4	
	3.2	2.2	3.2	3.6		3.9	3.4	2.1	2.5	1.2	
	3.4	1.8	2.5	3.7	5.4	3.3		0.8	3.5	1.5	
	2.6	1.8	2.8	4.3	5.2	4.6	1.7		2.7	1.2	
$\bar{X}$	3.2	2.0	3.7	4.1	3.5	3.1	1.8	3.1		1.3	$\bar{\bar{X}}$ 2.92
Biosorb absorbable powder 6 sterilizations	1.8	1.7	3.2	3.7	4.8	3.6		2.8		1.2	
	2.1	1.8		3.9	4.6	6.1	1.7	1.3		1.2	
	2.2	1.3	2.6	4.7		2.2	1.5	1.8		1.3	
	2.2	1.6	2.6	2.7	4.2	3.3	1.2	1.0		1.2	
	2.5	1.3	2.9	1.6	3.8			0.0	2.5	1.0	
$\bar{X}$	2.2	1.5	2.6	2.7	1.8	5.2	1.4			1.2	$\bar{\bar{X}}$ 2.17
	1.9		3.1	2.5	3.1	3.8	1.9		2.1		
	1.8	1.6	2.8	2.4	2.4	1.8	2.2	0.6	2.6	0.8	
Talcum powder 6 sterilizations	1.9	1.2	2.0	2.8	3.1	2.2	3.8	0.9	2.4	0.6	
	2.7	1.5	3.0	2.9	3.6	5.1	2.4	0.5	1.9	0.6	
	2.5	1.2	2.8	2.6			3.0	0.2	2.4	0.6	
$\bar{X}$	2.2	1.2	2.4	1.9	4.6	2.7		0.8	2.4	0.5	$\bar{\bar{X}}$ 1.93
Biosorb absorbable powder 9 sterilizations		1.3	1.9	2.3	2.5	3.3	0.9	2.3		0.6	
	1.5		2.3	2.0	2.6	1.8	2.0	0.9	2.0		
	2.0	1.1		2.4	2.6	2.4	1.9	0.0	1.1	0.8	
	1.6	1.0	1.7		2.7	2.2	1.9	0.0	1.7	0.7	
	1.6	1.6	1.6	1.6			2.4	1.0	1.7	0.7	
	2.2	1.1	2.0	1.8	2.7	2.3		0.8	2.0	0.6	
$\bar{X}$		1.1	2.1	2.3	3.0	5.8		0.9	2.3	0.7	$\bar{\bar{X}}$ 1.97
Talcum powder 9 sterilizations	1.9	1.2	1.4	2.1	2.1	6.5	2.1		1.8	0.7	
	1.6	1.7	1.8	1.9	2.8	4.2	1.7	0.6	2.3	0.3	
	1.6	1.2			3.1	4.9	2.7	0.6	1.9	0.5	
	1.7	1.2	1.6	2.0		2.8	1.7	1.1	1.4	0.5	
	1.8	0.7	1.8	2.3	3.1	4.6	3.2	0.6	1.5	0.5	
	1.7	0.9	1.6	2.4	2.8		2.3	0.5	1.4	0.6	
$\bar{X}$	1.7	0.9	1.7	2.7	2.8	7.4	1.8		1.7	0.4	$\bar{\bar{X}}$ 1.76
		1.1	1.6	2.2	2.7	5.0	2.1	0.7			
				1.7	2.7	2.0	1.6	0.5	1.2		
			2.3	2.8	3.8	1.8	0.6	1.0	0.5		
					2.1	1.6	0.8	1.5	0.3		
				4.2	1.8		0.5	0.6	0.4		
					0.6	1.1	1.0	0.2			
							0.3	0.3			

$\bar{X}$ = Average of five tests.

manner, the various data are comparable on a relative basis. The respective brands are designated by Roman numerals. An analysis* of the data in Table 1 indi-cates considerable variability among the tensile strengths of surgeons' gloves of various types and between gloves of a given manufacturer, even before prepara-tory dusting and sterilization. Initial ob-servation without the benefit of such an

analysis might create flash impressions as to the superiority of either lubricant but analysis shows that the gloves treated with the absorbable powder averaged 4 per cent stronger than gloves treated with talc, after three sterilizations. After six and nine sterilizations, Biosorb powder-treated gloves averaged about 12 per cent stronger than those treated with talc.

To determine the significance of the difference between the effects of the two lubricants on the degradation of tensile strength, a "t" test was made. The "t" value between the two lubricants computed on the thirty, paired, averaged, tensile strengths of groups of five samples is 3.16 with 29 degrees of freedom, indicating a highly significant difference in favor of the Biosorb powder. The interaction between the types of lubricant and types of fabric is insignificant, indicating that Biosorb powder retards the degrading effect of steam sterilization on the tensile strength of all three types of rubber.

With the brand of glove manifesting the least rapid rate of deterioration the fatigue point was not reached until the gloves had been subjected to nine autoclavings, regardless of whether talc or Biosorb powder was used as the lubricant. This fact is of considerable importance from the standpoint of operating room administration since there is considerable variation among all types of gloves in the inherent susceptibility of the material to autoclaving and adverse rates of deterioration are by no means necessarily attributable to the lubricating powder used in preparing the gloves.

This study statistically indicates a betterment in glove life and reliability by the use of Biosorb powder and it is clear on empirical grounds that this powder does not produce a more rapid rate of deterioration in comparison with that which occurs during steam sterilization when talc is used as the dusting agent. Thus, the factor of adverse effect upon rubber gloves is not encountered in avoiding the obvious hazards of talcum powder when this absorb-

able modified starch is used as the replacing agent.

SUMMARY AND CONCLUSIONS

1. A series of rubber gloves obtained from six manufacturers were divided into two sets and subjected to a comparative series of autoclavings after treatment, respectively, with talcum and a new absorbable starch powder.

2. The respective rates of deterioration were compared; it was found that when the absorbable powder was used the degradation in the tensile strength of the gloves produced by autoclaving occurred less rapidly upon repeated sterilizations than when talcum was used as the dusting agent.

3. The effect of this absorbable powder on rubber gloves does not constitute a drawback to its use as a replacement for talcum powder as a lubricating agent.

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CERTAIN living tissues will grow in a medium consisting of Tyrode's solution, buffered salt solution, heparinized plasma and embryonic extract. Neither the plasma nor the embryonic extract need come from the same species of animal.

From "Principles and Practice of Surgery" by W. Wayne Babcock (Lea & Febiger).

SURGICAL COMPLICATIONS OF INTESTINAL TUBERCULOSIS AS SEEN AT NECROPSY*

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IT is well known that the incidence of intestinal tuberculosis is high in known pthisic patients who are seen at autopsy. Various authors[1-3] have reported percentages that vary from 50 to 90 per cent. Blumberg[4] in his study on the inci-

INCIDENCE OF PULMONARY AND INTESTINAL TUBERCULOSIS

There have been no reports in recent years on the incidence of pulmonary tuberculosis and its intestinal complications in general hospitals. A review of 4,200 consecutive autopsy protocols at Morrisania City Hospital from 1930 to 1946 revealed an incidence of 4.05 per cent (170 cases) of active pulmonary tuberculosis. In this group of 170 pthisic patients there was an incidence of intestinal tuberculosis of only 28.23 per cent (forty-eight cases). The sites of predilection were the ileum and cecum. (Table I.)

The figure of 28.23 per cent is considerably lower than that reported in the current literature[3] and text books;[1,2] however it must be emphasized that the material in this series comes from a general hospital. Our patients with tuberculosis are transferred as expeditiously as possible to tuberculosis institutions once the diagnosis is made. This state of affairs naturally tends to limit our patients to those that were unsuspected prior to admission and to those who were acutely ill and could not be transferred.

Because of this situation one would suspect that the incidence of complications in intestinal tuberculosis would tend to be higher in institutions such as ours despite the smaller number of cases. This point

TABLE I
FORTY-EIGHT CASES OF INTESTINAL TUBERCULOSIS

Location of Lesions	Number	Percentage
Jejunum	5	5.87
Ileum	26	30.59
Upper	5	
Lower	21	
Cecum	18	21.19
Appendix	2	2.35
Ascending colon	9	10.59
Transverse colon	6	7.06
Descending colon	6	7.06
Sigmoid colon	4	4.70
Rectum	2	2.35
Diffuse involvement of small and large bowel	7	8.24
Total	85	100%

dence of intestinal tuberculosis, classified the pulmonary lesion as seen at autopsy into far advanced, moderately advanced and early cases. He then found that the incidence of intestinal tuberculosis varied markedly with the severity of the pulmonary lesion; 70 to 80 per cent of the far advanced pulmonary cases showed intestinal involvement, 14 to 18 per cent of the moderately advanced and only 5 to 8 per cent of the early cases showed involve-

* From the Department of Pathology, Morrisania City Hospital, New York, N. Y.

was brought home to the authors who saw at necropsy in a relatively short period of time a case of intestinal obstruction and another of generalized peritonitis due to perforation of the small bowel; both cases being secondary to intestinal tuberculosis.

TABLE II

FORTY-EIGHT CASES OF INTESTINAL TUBERCULOSIS

	Number	Percentage
Perforated with generalized peritonitis	5	10.42
Site of Perforations		
Ileum	3	
Sigmoid	1	
Small and large bowel (multiple)	1	
Feces in peritoneal cavity	2	
Age range—15 to 66 years {Male	2	
{Female	3	
Operated ante-mortem	3	
Intestinal Obstruction	6	12.50
Adhesions	5	
Stenosis secondary to scar tissue	1	
Age range 21-77 years {Male	2	
{Female	4	
Operated ante-mortem	3	

complete or incomplete perforation. Cullen[3] in a review of over 700 cases of intestinal tuberculosis seen at necropsy at Seaview Hospital found that only 3.81 per cent had perforations. While our series is small, the fact remains that over a period of sixteen years 10.42 per cent of our patients with intestinal tuberculosis seen at autopsy had complete perforation with generalized peritonitis. The current foreign literature[16-18] contains case reports which serve to emphasize this fact.

PERFORATION IN INTESTINAL TUBERCULOSIS

Some authors[5,6] lay great stress on the danger of intestinal obstruction in ulcerative intestinal tuberculosis if and when it heals. The question of acute perforation with generalized peritonitis is not stressed. The consensus of opinion[2,5,7,8,9,15] is that perforation is uncommon; if it does occur, the process is a slow one with a localized peritoneal reaction and a walling-off of the perforation.

The criteria for the diagnosis of acute perforation of the bowel at necropsy were: (1) Demonstration of the perforation and (2) generalized peritonitis with either feces and/or pus in the peritoneal cavity. Five patients out of our forty-eight cases of intestinal tuberculosis met these criteria, an incidence of 10.42 per cent. (Table 11.)

The figure of 10.42 per cent is significantly higher than the incidences for perforation reported from tubercular institutions. Brown and Sampson[10] found that only 3.7 per cent of their patients had

OBSTRUCTION IN INTESTINAL TUBERCULOSIS

Boyd,[1] Portio[7] and Barrow[11] all believe that the most common cause of intestinal obstruction in intestinal tuberculosis is adhesions rather than stenosis secondary to cicatrization following the healing of the intestinal lesions. Brown and Sampson[10] in their book on intestinal tuberculosis state "stenosis is infrequent in secondary intestinal tuberculosis as the patient usually dies before sufficient healing takes place."

There were six patients in our series of intestinal tuberculosis that showed intestinal obstruction, an incidence of 12.50 per cent. Five of these cases were due to adhesions and one to stenosis. (See Table 11.) This latter patient was a fifty-two year old male who had known pulmonary tuberculosis for twenty years. Barrow[11] in reporting on sixty-seven cases of tuberculous peritonitis found that nine of them were operated upon for intestinal obstruction due to adhesions only. This is an incidence of 13.43 per cent which compares favorably with the incidence of 12.50 per cent obtained at autopsy in this series.

PRIMARY INTESTINAL TUBERCULOSIS

There have been reports[12,14] in recent literature of primary tuberculosis of the intestines; however, most authors[7,8,9,12,13,14] are agreed that although primary tuberculosis of the intestines can occur it is extremely rare. Bockus[8] states that the literature contains very few instances of

primary ulcerative intestinal tuberculosis in persons beyond eight years of age. He lays down the following criteria for diagnosis: (1) Characteristic histological picture; (2) demonstration of acid-fast bacilli in the intestinal lesion; (3) absence of tuberculosis in other parts of the body and (4) transference of the infection to laboratory animals.

Crohn and Yarnis[14] in an excellent review of the subject laid down similar criteria for diagnosis. These authors surveyed all the autopsies (4,800) and surgical material at Mt. Sinai Hospital from 1926 to 1938. Among the necropsies they found only one case and among the surgical material only seven cases that they felt were primary intestinal tuberculosis.

Among our 4,200 necropsies, we found only one case that may be considered as one of isolated or primary tuberculosis of the intestines. Because of the rarity of this condition, we wish to present the case report* and autopsy findings in brief.

CASE REPORT

A sixty-six year old white male was admitted to Morrisania City Hospital on April 26, 1946, and died on April 28, 1946. The patient had been in this hospital several times since 1936 for a fractured rib, fractured humerus and chronic alcoholism.

He was admitted complaining of abdominal pain and vomiting of five days' duration. The pain was knife-like and peri-umbilical at the beginning but it gradually shifted to the lower abdomen. The vomitus was "brownish material." He had passed some flatus and a watery stool on the day of admission. There was no history of diabetes, tuberculosis or cardiac disease. The patient had lost 20 to 30 pounds in the past year. There were no other gastrointestinal complaints.

The physical examination revealed the following positive findings. Temperature 101.2°F., pulse 120, respirations 40 and blood pressure 114/70. The patient was relatively comfortable. The abdomen was tympanitic and there was generalized rigidity, tenderness and rebound.

* From Surgical Service of Dr. Joseph Deutsch, Morrisania City Hospital.

These findings were most marked in the right upper and right lower quadrants. Rectal examination revealed tenderness above and lateral to prostate. The blood count was white blood cells 5,150, polymorphonuclears 59 per cent, lymphocytes 38 per cent and monocytes 3 per cent. An x-ray of the abdomen in the upright position revealed air under the diaphragm.

The patient was sent to surgery for an exploratory laparatomy. His postoperative temperature ranged from 101° to 103°F. and his pulse was from 90 to 120. He developed cardiac arrthymia which became normal after digalin therapy. On the second postoperative day the patient developed pulmonary edema and made an exodus.

At necropsy the patient was found to have a perforation in the ileum and generalized peritonitis. When the ileum was opened a sharp stricture was found about 3 feet proximal to the ileocecal valve. At this point there were several small ulcers girdling the bowel which was thickened and firm. Spreading proximally to this for a distance of 4 cm. was a softer, more red and acute looking, sharp-edged ulceration that had a perforation. The rest of the organs were normal.

Microscopic sections through the perforated ulcer revealed the typical histopathology of tuberculosis. Acid-fast organisms were demonstrated in the microscopic sections. Unfortunately no animal inoculations were done. No evidence of active tuberculosis or open lesion was found by x-ray or at autopsy.

The presumptive diagnosis in this case is primary tuberculosis of the ileum with perforation and generalized peritonitis.

SUMMARY AND CONCLUSIONS

1. Review of 4,200 consecutive autopsy protocols in a general hospital revealed the incidence of pulmonary tuberculosis to be 4.05 per cent (170 cases).

2. The incidence of intestinal tuberculosis among the active pulmonary tuberculous cases was 28.23 per cent (48 cases).

3. The most frequent site of involvement in intestinal tuberculosis is the lower ileum and the cecum.

4. The incidence of intestinal perforation with generalized peritonitis in intestinal tuberculosis was found to be 10.42 per cent

which is much higher than has been reported from tuberculosis institutions.

5. The incidence of intestinal obstruction in intestinal tuberculosis was 12.50 per cent. The most common cause of intestinal obstruction is adhesions and not stenosis secondary to cicatrization.

6. The incidence of surgical complications (perforation and obstruction) in intestinal tuberculosis in a general hospital is high (22.92 per cent) despite the small number of patients seen.

7. A case history and necropsy findings of a case of primary tuberculosis of the ileum with perforation and generalized peritonitis was presented.

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Case Reports

TRANSPYLORIC HERNIATION OF REDUNDANT GASTRIC MUCOSA

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IN an investigation concerning mechanical causes of so-called "gastric ileus," Meyer and Singer,[1] in 1931 noted that von Schmieden,[2] twenty years earlier, had fully described a case of gastric mucosa prolapsing into the duodenum. A partial translation of this case report was presented in their paper. In von Schmieden's words, "Such a finding up to now has not been described in the literature."

In 1926 Eliasen, Pendergrass and Wright[3] reported two similar cases. They stated that these "are the first two cases that have been reported to our knowledge." This must have been a slight slip, not because von Schmieden's case had been overlooked; the latter, hidden as it is in an extensive publication, is not easy to find even with the correct reference. However, Eliasen and Wright,[4] in 1925, described fully a similar case which appears not identical with any of the two cases in their subsequent report.

Since then several reports have appeared. namely: by Meyer and Singer,[1] one case (1931); Pendergrass and Andrews,[5] three cases (1935); Rees,[6] four cases (1937); Bohrer and Copelman,[7] one case (1938); Archer and Cooper,[8] four cases (1939); Melamed and Hiller,[9] one case (1943); and Norgore and Schuler,[10] two cases (1945).

In a discussion of non-malignant lesions of the stomach, Eusterman and Balfour[11] (1935) briefly mention two cases which apparently fall into the category of the present survey although no detailed presentation is given.

In a recent report, Scott[12] describes his experience from a large Naval hospital, where, in 1,346 roentgen examinations of the upper gastrointestinal tract a prolapse of the gastric mucosa was found in fourteen patients, that is, 1.04 per cent. Interestingly, "In this group of young and adult men, prolapse of gastric mucosa occurred as often as gastric ulcer."

We have made the diagnosis of prolapsing gastric mucosa in seven patients in some 800 successive gastrointestinal x-ray examinations. In addition, a minimal prolapse of redundant gastric mucosa was present in six patients with gastric ulcer.

It is somewhat unexpected that since von Schmieden's original report, the subsequent publications concerning this condition all have appeared in American literature only. One has the notion that subsequently others may be pointed out and previously overlooked because of non-specific titles.

From this survey one is left with the impression that in spite of the relatively few cases reported, this type of gastric pathologic condition may prove not as uncommon as one might have been led to believe. With increasing knowledge of the characteristic roentgenologic appearance of prolapsed gastric mucosa, it seems probable that some patients previously listed under the headings of gastric neurosis or psychosomatic disorder, upon re-examination may be found to fall into the group of organic gastric disorders. Without the knowledge of even the existence of such a

condition, we, in our own limited experience, would not have been prevented from making the incorrect diagnosis of functional disorder in two instances at least.

In the case reports a varying terminology is employed, namely, herniation, intussusception, prolapse or extrusion of gastric mucosa through the pylorus. Although the term extrusion may be the more correct form, as pointed out by Norgore and Schuler,[10] use of the other terms would not be likely to lead to confusion as to the actual condition, namely, the existence of a redundant fold or folds of the antral gastric mucosa protruding through the pylorus into the duodenum.

Unexpectedly, inaccuracies are noted in the literature due to inclusion of herniated "gastric mucosal lesions," that is, benign or malignant tumors. The similarity in anatomic location and mechanical peculiarities resulting therefrom, seem not sufficient to consider them under the same heading with true antral mucosal extrusions.

Before proceeding with a further discussion of this condition, the following report may be of interest:

CASE REPORTS

CASE 1. The patient was a Norwegian, forty-five years of age, who had worked as a seaman throughout his adult life. An older brother had been operated upon, allegedly for "stomach ulcer." A younger sister had been medically treated for a "duodenal ulcer."

The patient had had "influenza" during the flu epidemic of 1918. In 1934 he had been successfully operated upon for left-sided inguinal hernia.

The onset of his present condition dated back to 1933 when he suffered from dyspepsia for a period of three months. Since its onset the distress has been intermittent. Episodes of distress of a couple of weeks up to eight months' duration would alternate with periods of reasonable freedom from distress. Particularly distressing periods had been present in 1933, 1934, 1937, 1941, 1942, 1944 (for ten months) and from November, 1945, to the time of admission to White Plains Hospital.

The subjective symptoms during periods of distress had not essentially changed in character over the years, excepting an increased intensity of the distress and longer-lasting distressing periods. The patient volunteered the suggestion that the aggravation of late may have resulted from the nervous tension aboard ship during World War II, with ever lurking danger of torpedoeing, mines and bombing.

The distressing periods would be characterized by epigastric fullness and distention, with griping non-radiating pain in the midepigastrium, followed by frequent eructation, nausea and vomiting. The distress would frequently come on immediately after or while eating. It would instantly be improved upon spontaneous or induced vomiting. It never occurred at night. The patient never suffered from hunger pain. He had not experienced any particular relief after the intake of alkalis. He was under the impression that during the active periods of distress it made no difference what kind of food he ate; even water would then frequently bring on pain and vomiting.

On several occasions the patient had had hematemesis, followed shortly by the passage of tarry stools for a few days. Hematemesis and melena had been noted for the first time in 1933; since then repetition of these phenomena had occurred in 1937, 1942 and 1944. Following the gastrointestinal hemorrhage in 1944, the patient was investigated at a hospital elsewhere, during which time no definite cause of the gastrointestinal hemorrhage could be ascertained. The patient convalesced for eight months before signing aboard ship again.

In November, 1945, his subjective distress recurred. Because of sickness he signed off his ship in New York at the beginning of January, 1946. This incident was duly celebrated by what was termed a moderate spree. On January 6, 1946, following profuse vomiting of blood, the patient collapsed and was admitted to a hospital while unconscious where he remained until February 12, 1946. Investigations failed to reveal the definite cause of the bleeding. Roentgen rays of the gastrointestinal tract did not reveal the presence of ulcerations or tumors. However, it was observed that there was pronounced prominence of the gastric rugae. After the effect of the acute hemorrhage had been overcome by blood transfusions, the patient was started on a dietary medical regime. On February 4, 1946, a hemorrhoidectomy was

FIG. 1. For comparative purposes the normal pyloric canal, the normal duodenal bulb and normal mucosal pattern of the bulb are illustrated in two normal cases, A and B.

FIG. 2. Case 1. Serial studies of the duodenal bulb under compression with patient in the erect position. Characteristic gastric folds which appear to be continuous with the prepyloric folds are seen within the base of the duodenal bulb.
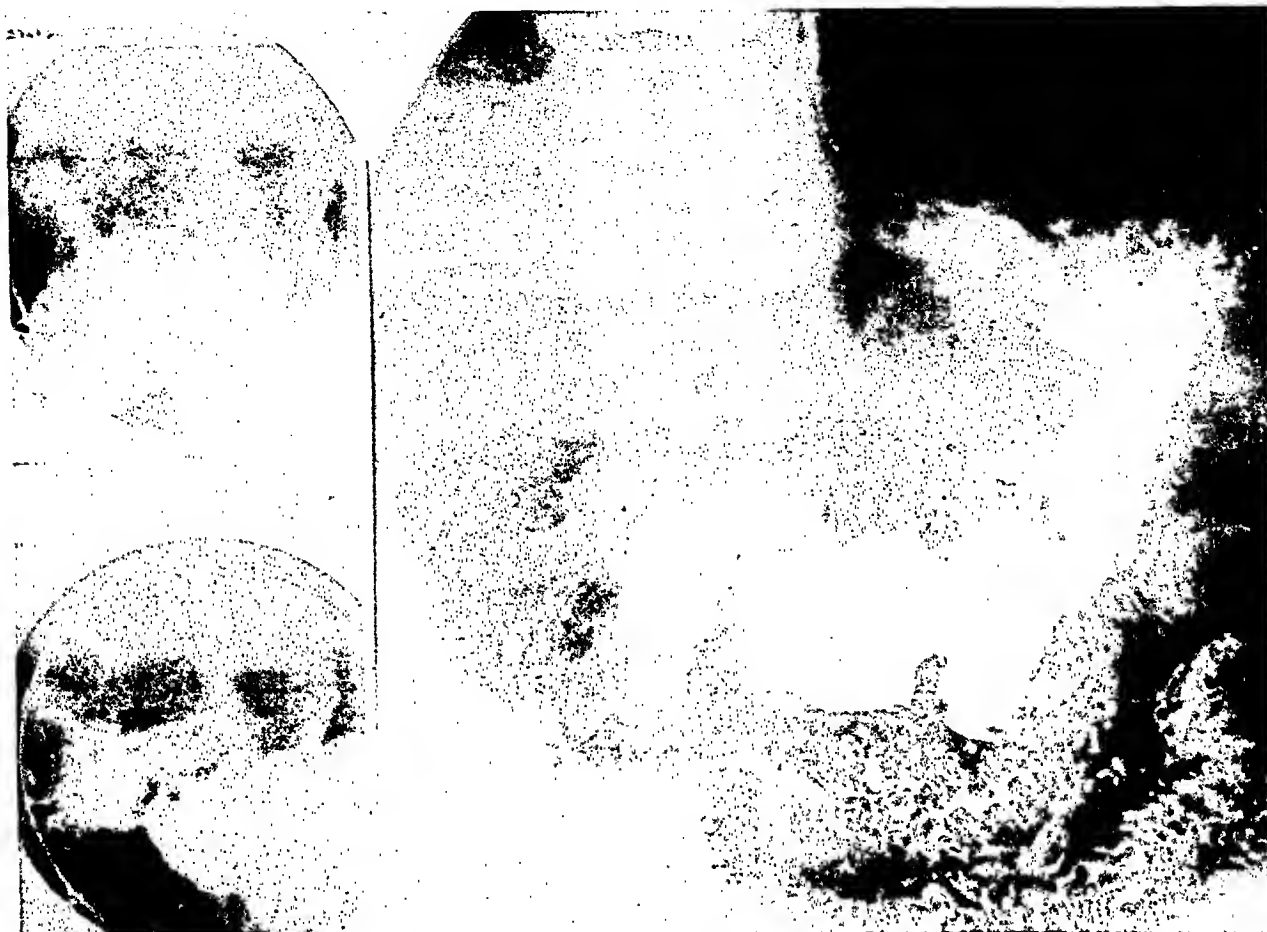
Fig. 3. Case 1. The wide open pyloric canal and the changing pattern of the prolapsed gastric mucosa, coincident with the antral systole and diastole. In the survey film on the right the large gastric rugal pattern can be recognized.

likewise performed. After his dismissal the patient continued the dietary regime at a nursing home.

In spite of a prolonged convalescence with a carefully supervised dietary regime, the patient continued to complain of frequent epigastric distress and vomiting; there was no further recurrence of the gastrointestinal hemorrhage. The patient therefore was referred to the Norwegian Public Health Service, New York, for further investigation. X-ray investigations were performed by one of the authors, Dr. Lewitan, on two different occasions and revealed the following: (Figs. 1, 2 and 3.)

The esophagus was fluoroscopically negative. Stomach was normal in size and position. There was a moderate amount of secretion present at the onset of the examination. The stomach walls were completely pliable. The gastric rugae appeared to be considerably enlarged. There was no evidence of gastric ulceration or other intrinsic pathologic symptom. The pyloric opening was immediate and the pylorus appeared to be wide open. The gastric emptying time was increased. Within the base of the duodenal bulb, on moderate compression, translucent bands were apparent which were continuous with the prepyloric gastric rugae. There was scalloping at the apex of the bunched-together prolapsed gastric folds. The contour was constantly changing and the relative volume of the prolapsed gastric folds appeared to increase with antral systole. In antral diastole the gastric folds were seen in the juxtapyloric region. The base of the duodenal bulb appeared unusually broad. Even in antral systole the pyloric ring appeared very wide. At times the resemblance to a small cauliflower was pronounced. The findings were present with the patient in the erect as well as recumbent positions. In supine views it was absent. No other significant findings were present on roentgenographic and radiographic examinations of the remaining gastrointestinal tract. The same findings were obtained on the second examination after the patient had had a renewed medical regime from March 12th to April 20th. The only significant change seemed

a slight regression in the general caliber of the gastric rugae.

On May 5, 1946, the patient was admitted to White Plains Hospital. He was in good nutritional status, of the stocky type, with no history of weight loss and weighed 183 pounds. His height was 5 feet, 8 inches. His blood pressure was 115 mm. Hg systolic and 80 mm. diastolic. A carefully performed physical examination revealed no demonstrable pathologic findings except slight tenderness to deep pressure in the area of the mid-epigastrium. Laboratory examinations revealed 4,700,000 erythrocytes per cu. mm. with 13.4 Gm. hemoglobin; leukocytes numbered 7,000 with normal differential count. Plasma proteins were 6.4 Gm. per cent; chlorides, 620 mg. per cent. Stool examination did not indicate the presence of occult blood.

Following the Ewald test meal, at the end of one hour no free hydrochloric acid was found, and the total acidity was 15 units.

Following preoperative preparation, the patient was submitted to an operation on May 9, 1946, by Dr. Nygaard; this was performed under continuous spinal anesthesia.

A careful investigation of the intra-abdominal organs revealed no demonstrable pathologic condition excepting that pertaining to the stomach and duodenum. The duodenum was free of adhesions, deformity or palpable induration of the anterior or posterior duodenal wall. There were no indurations of the head of the pancreas and no adhesions between the posterior aspects of the stomach and the peripancreatic capsule. On the basis of the preoperative x-ray diagnosis, particular attention was directed toward the pyloric and prepyloric areas. Careful inspection of this area did not reveal any demonstrable pathologic disease; in particular there were no irregularities of the anterior wall nor any visible bulging areas. Finger palpation at first gave the impression of a peculiar thickening of the pyloric segment. By "milking" the prepyloric area with one finger anteriorly and one posteriorly, one had the sensation that this thickened area could be moved up and down. By slowly moving the two compressing fingers from the prepyloric area in distal direction, the sensation of an elastic induration would abruptly cease about 1.5 to 2 cm. distal to the pyloric vein. By then moving the fingers again slowly in a proximal direction, the first part of the duo-

denum and the pyloric ring would feel perfectly normal, while the prepyloric area again would give the sensation of fullness. One was also struck by the fact that while at one moment finger palpation gave the impression of a certain, definite configuration of the elastic induration, during the next moment this may have changed or disappeared. The remainder of the stomach appeared normal.

As is obvious also from the several reports in the literature concerning the surgical treatment to be chosen, the surgeon is confronted with a decision as to the procedure preferable in the individual patient. In the subject herein described we believed that the repeated and occasionally very severe episodes of gastrointestinal hemorrhages weighed heavily in favor of a partial gastric resection; the latter procedure was therefore adopted, removing about 70 per cent of the stomach. The remaining segment of the stomach was joined with the jejunum, employing a procedure ad modum Polya.

The following is the report of the pathologist from a gross specimen of the stomach. Specimen consists of the major portion of a stomach; it measures 25 cm. along the greater curvature and 9 cm. along the lesser. Specimen includes the pyloric ring and approximately 1.5 cm. of the duodenum. The outer surface bears a few fibrous and fatty tags on its posterior wall and there is attached a small amount of omentum and of the greater hepatic ligament. The serosa otherwise is smooth and glistening. The wall is well developed and when the organ is opened the mucosa is found to present somewhat unusually high, prominent folds throughout. The mucosa averages 1 mm. in thickness and beneath it there is a quantity of loose submucosa that, in the folds, attains a thickness of 1 cm. Immediately at the pylorus the mucosa is even more redundant than elsewhere and the pyloric ring consists of a well developed fold of mucus membrane 1 cm. high overlying a quantity of submucosa and a continuous muscular ridge 5 mm. in diameter. The loose mucosa at the pylorus can be slipped easily over the ring so as to cover entirely the portion of the duodenal mucosa. The mucosa of the latter is grossly normal.

Just proximal to the pyloric ring the posterior wall of the stomach near its mid-point presents a shallow linear defect that runs longitudinally a distance of 8 mm. and has a maximum width

of 2 mm. The edges of this are smoothly rounded and the base is made up of submucosa. There is no injection or hemorrhage about this defect, nor is there evidence of scarring. The opposite edges of the defect can be easily approached. Elsewhere the mucosa is intact; there are a few small bright red hemorrhages into it. The muscularis except for the hypertrophy of the pyloric ring, is only slightly heavier than normal, averaging 1.5 mm. in thickness.

Histologic findings are reported as follows: The mucosal architecture is regular and the glands are of the usual type, but here and there the superficial portion of a gland is dilated and is lined by high columnar epithelium of the intestinal type. Small papillary infoldings have been formed within several of these glands. The tunica propria is normal in amount but at many places there are dense infiltrations of lymphocytes, together with a few plasma cells and large mononuclear cells. At several points these infiltrations take the form of lymphoid follicles with ill defined germinal centers. Plasma cells predominate in one block from the pyloric region.

Two blocks taken through the linear defect noted just proximal to the pyloric opening show only a shallow V-shaped infolding of the mucosa, completely lined by glands. The glands here run somewhat irregularly and there is an increase of fibrous tissue in the tunica propria. It probably represents a healed erosion. The muscularis mucosae and the submucosa beneath it are normal.

Diagnosis: Chronic gastritis, redundant gastric mucosa, prolapse of gastric mucosa through pylorus and erosion of stomach, healed.

The patient tolerated the procedure very well; the postoperative course was uneventful. From the fourth postoperative day he was able to enjoy intake of fluids and diet by mouth, the diet being gradually increased and tolerated well. The patient was dismissed in good general condition on May 30, 1946. Following one month of further convalescence, he again signed aboard ship.

Etiology. In considering the origin of this condition one is at a disadvantage due to insufficient information regarding the actual pathologic picture present. True enough, in many of the reports pathologic investigations of the excised mucosal folds

are presented. In others, such is lacking. In the two previous cases,[10] as well as in our present patient in whom partial gastric resection was performed, a diffuse chronic gastritis was noted. The presence of a chronic gastritis, however, has not been noted in some of the subjects with local excision of the mucosal folds. Reversely, we have had occasion to verify the absence of mucosal prolapse in cases of generalized hypertrophic gastritis of a most pronounced degree. Extrusion of antral gastric mucosa is not necessarily a characteristic part of the x-ray picture in that condition.

Eliasen and Wright,[4] in a discussion of the etiology of benign polyps of the stomach, have visioned a development from local mucosal hypertrophy to polyp formation as resulting from peristaltic action. The local hypertrophy would originate in a low grade mucosal inflammation due to physical, nutritional, functional, chemical or bacterial factors. According to this view, chronic gastritis in due time may lead to adenoma which may finally undergo malignant transformation.

In three operated patients Rees[6] mentions the finding of a constricted pyloric ring which, upon transection, appeared as a "white fibrous ring, both smaller and thinner than normal pyloric muscle and with no elasticity." He is of the opinion that this represents the primary pathologic condition and that, secondarily, hyperperistalsis of the stomach would lead to loosening of the mucosal attachment to the muscularis and finally to extrusion. Other investigators, however, have failed to note a similar pyloric muscle constriction. The significance of these observations, furthermore, seems uncertain in the absence of biopsy of the transected pyloric ring.

On the basis of normal anatomy of the various layers of the stomach wall as well as the physiologic peristaltic action of the gastric mucosa as well as the entire stomach wall, Scott[12] reasoned that "the structural conditions necessary for the development of a prolapse are inherent in the walls of the normal stomach. A prolapse occurs

only after the fibers in the flexible submucosa have been stretched and loosened by abnormal gastric peristalsis which, in turn, is initiated by neurogenic or chemical stimuli or a combination of both."

Symptomatology. A study of the reported histories in patients with extrusion of antral gastric mucosa gives one the impression of a diffuse type of dyspepsia. At least at present, on the basis of available information, it appears that there is no one symptom or grouping of symptoms or signs that may justify a definite clinical diagnosis without the aid of roentgenologic investigations.

Abdominal distress appears to be present in practically all of the subjects. Eructation, bloating and sensations of fullness after meals may be the only distress. In others may be noted intermittent episodes of cramp-like pain localized to the epigastrium or right upper abdominal quadrant, frequently aggravated by the intake of food, particularly heavy food. The intake of alkali is often found to be of little consequence. Several patients noted some benefit from soft or liquid diet. A considerable loss of weight is not uncommonly encountered.

In several of the patients a moderate gastric residue has been noted as determined by x-rays.[1,5,12] In no patient, however, can it be seen that gastric dilatation has occurred secondarily to complete pyloric obstruction resulting from prolapsed antral mucosa. Likewise, no case has been reported in which one can speak of a "gastric ileus" simulating the ball-valve syndrome caused by sudden invagination of a prolapsed polyp or neoplasm or foreign body, leading to a severe emergency condition requiring immediate surgical intervention.

Gastrointestinal bleeding is not a rare complication in these subjects.[5,7,8,12] Gastrointestinal bleeding may appear as intermittent occult blood in the stool only and may or may not be associated with secondary anemia. It may, however, present itself as acute episodes with profuse bleeding, resulting in collapse, as occurred in our patient. It is probable that bleeding arises from gastric mucosal erosions on a traumatic basis similar to those noted in certain cases of diaphragmatic hernia.

In our own patient, in whom partial gastric resection afforded full opportunity for close scrutiny of the removed part of the stomach, one was struck with the fact that an innocent appearing erosion was the only positive finding that could explain the previous recurrent, severe episodes of bleeding. Whether or not actual ulcerative processes may occur in the prolapsed mucosa remains uncertain. The ulcerations as described in one report[9] lose significance, as it appears from the operative as well as the pathologic report that one was dealing not with simple antral mucosa extrusion, but with a pedunculated fibroma projecting through the pylorus.

In this connection it may be pointed out that malignant degeneration in a true case of antral mucosal prolapse is as yet not reported. The frequent reference to such a possibility is based on a report[12] which, by closer study, proves to deal with a generalized gastric polyposis with malignant degeneration.

From what has been stated, it seems evident that extrusion of antral gastric mucosa must be considered in the differential diagnosis in patients with obscure episodes of gastrointestinal hemorrhage. Retrospectively, this has forcefully been brought to our mind when reviewing the history of one of our subjects observed in 1944.

CASE II. The patient was a seaman, aged fifty-one years, who had been perfectly well up to the spring of 1944 when he was forced to go ashore because of increasing shortness of breath and weakness. There was no history of dyspepsia prior to the onset of this disease, nor did such appear to any great extent during the one and a half years he remained ashore before his repatriation to Norway at the end of the war. His only abdominal complaint was occasional epigastric fullness and eructations after meals. He was a moderately heavy drinker. Laboratory tests revealed a pronounced secondary

FIG. 4. Case II. Prolapse of the antral gastric mucosa into the base of the duodenal bulb.

anemia. Gastric acidity tests revealed values within normal limits for total and free hydrochloric acid. Repeated stool examinations at repeated intervals over a long period of time proved the constant presence of occult blood in the stool. During the time the patient remained under observation, an endless number of investigations accumulated and a series of consultations were held. On five different occasions a complete gastrointestinal investigation was performed by three different roentgenologists. No definite lead was ever secured as to the source of the occult blood in the stool. Over this period of one and a half years, the patient was given in all, eight blood transfusions in addition to intensive anti-anemic medication.

The patient was reluctant to accept the proposal of exploratory laparotomy as he "did not feel sick, only somewhat weak at times," when his hemoglobin fell below 60 per cent. Our own uncertainty as to the source of the bleeding as well as the uncertainty of detecting this by an exploratory laparotomy, admittedly did not constitute a too convincing basis for overcoming this intelligent patient's objection to an operation.

The case was taken up for review after we had operated upon the patient whose history is detailed earlier in this paper. This was several months after the repatriation of our patient. The review again brought out that hypertrophic gastritis had been reported in each of five gastrointestinal x-ray investigations. It was with no small chagrin that we then noted that in two of the x-ray series undertaken by one of the authors, Dr. Lewitan, a correct description had been given of a prolapsed gastric mucosa visible in the x-ray pictures of both series. (Fig. 4.) In spite of it being observed as well as reported, at the time we considered this merely a coincidental finding. Subsequent clinical experience indicates that in all probability we did grossly err in this assumption.

One cannot with absolute certainty, state that this review of the case identifies the extrusion of antral mucosa as the source of the bleeding, inasmuch as no operation was performed. This, however, does not change the following important practical point: With the roentgenologic demonstration of mucosal prolapse, coupled with a realization that this may give rise to a
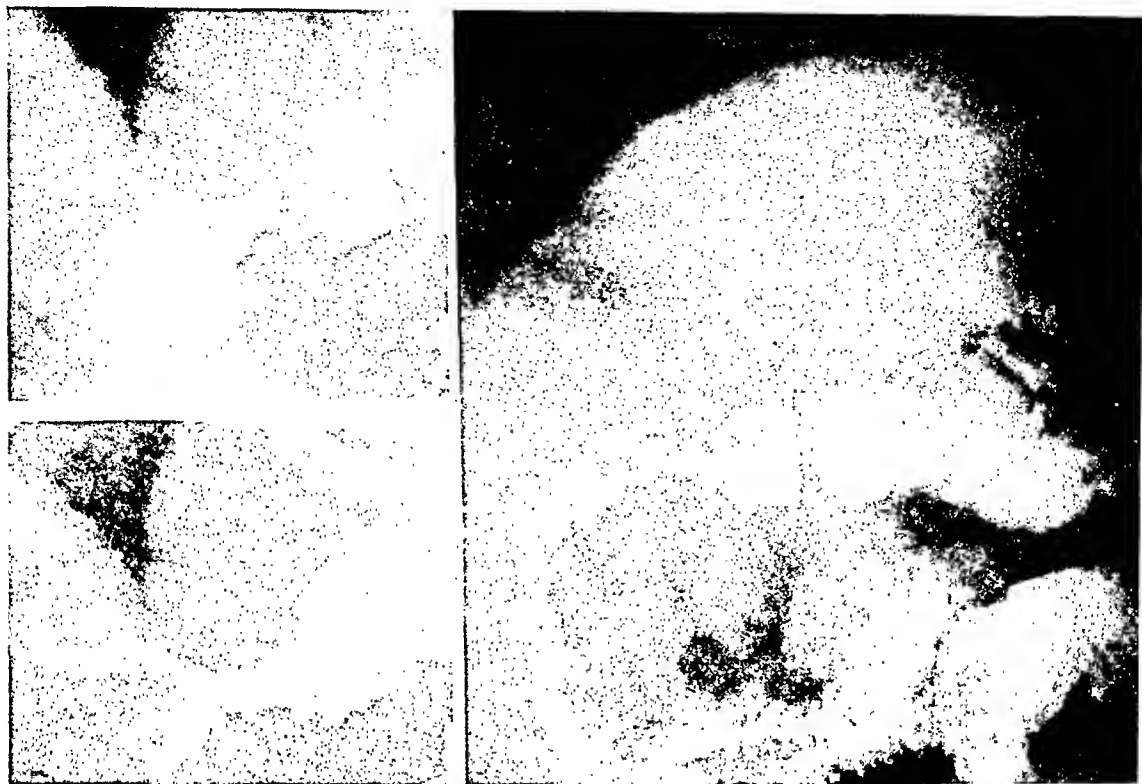
FIG. 5. The appearance in minimal prolapse; wide open pyloric canal and translucent defect with striations in the base of the bulb. At the apex of the defect there is scalloping.

gastrointestinal hemorrhage, a proposed surgical intervention would thereby have been given a definite diagnostic basis as well as an important cue for the surgical approach. We failed, not in the x-ray demonstration of the prolapse, but in the realization of the various clinical pictures that may arise from such a condition. The object lesson seems clear. The whole responsibility cannot be made to rest with the roentgenologic department only. Evaluation of the findings rests with the clinician.

In the x-ray examination of a suspected lesion of this kind, certain technical details have to be observed. Pendergrass and Andrews[5] stressed prone views as essential. The patients included in this report showed gastric mucosal folds in the duodenum in the erect as well as in prone positions. Overdistention of the stomach by a great amount of barium, however, would completely obscure the presenting pathologic symptoms as well as supine views. Compression or incomplete filling would bring the pathologic condition out most clearly. (Fig. 2.)

Radiographically, the following features are of diagnostic importance: A filling defect in the base of the bulb resembling a small cauliflower. The defect permits recognition of characteristic gastric folds by the presence of striations and scalloping. The gastric rugae in the duodenal bulb are continuous with the prepyloric folds. The defect changes its size and volume coincident with antral systole and diastole. The pylorus appears wide open and gastric emptying time is increased. There is usually an increase in the caliber of the gastric rugae and the presence of hypersecretions at the onset of examinations is also noted. (Figs. 5, 6 and 7.) Once the roentgenologist is trained to evaluate correctly the changing pattern of prolapsing redundant gastric mucosa, the diagnosis will be made more frequently. The demonstration of minimal prolapse may be looked upon as objective evidence of the presence of a gastritis, for which definite

diagnostic criteria roentgenologically are still lacking. A demonstration of the small prolapsing lesions in association with gastric ulcer is not surprising because of the frequent association of gastritis with gastric ulcer. (Fig. 8.)

Treatment. With our hypothetic understanding of the origin of antral mucosal extrusion at present, medical treatment necessarily will be essentially of a symptomatic character. As pointed out, cases are reported in which co-existence of gastritis was noted. When such a condition is noted to be present, however, medical treatment as for gastritis would be the logical approach. Definite improvement has been noted after a bland diet with frequent small meals. Antispasmodics suggest themselves in view of the irritability of the pyloric ring resulting from mechanical stimulation by the pendulating movements of the mucosal folds.

In some patients antacid medication has been of no avail; in others it has given intermittent relief. In a patient recently under our care, best results were obtained by a moderate dose of milk of magnesia at night. Occasionally, rest after meals is found comforting because of a slipping back of an extruded fold into the antrum. In general, no fast rules as to the medical regimen can be laid down. Each patient requires careful individual consideration.

A patient has asked us if medical treatment would ever result in retraction of the mucosal folds. At present we are not in a position to answer whether or not this condition represents a reversible process. One of our patients has been carried on a medical regimen for two years. Repeated roentgenologic examinations during this period, at least up to the present time, have given no indication of any essential changes in the x-ray demonstration of the mucosal extrusion. In this patient the subjective moderate distress has likewise been practically stationary although never severe enough to make him discontinue his regular activities as a mechanic In a few other subjects the observation time has been

FIG. 6. The appearance of the prolapse in another of our patients at the first examination in 1944. With the patient erect the film shows the presence of hypersecretions which form a fluid level with the barium mixture.

shorter. Our impression is that in none of these has there been any gradual progression of the degree of extrusion. In one of these patients the history of dyspepsia of the same character as at present dates back to 1937. With some justification, therefore, it appears that a progressive enlargement of the prolapsed mucosal folds does not necessarily take place. Whether or not the process is reversible must await further observations.

As to the question of medical *versus* surgical treatment of this condition, the decision will rest upon an analysis of each individual patient.

Up to now, no case has been reported leading to acute gastric dilatation due to pyloric obstruction. On theoretic grounds only such a "ball-valve phenomenon,"
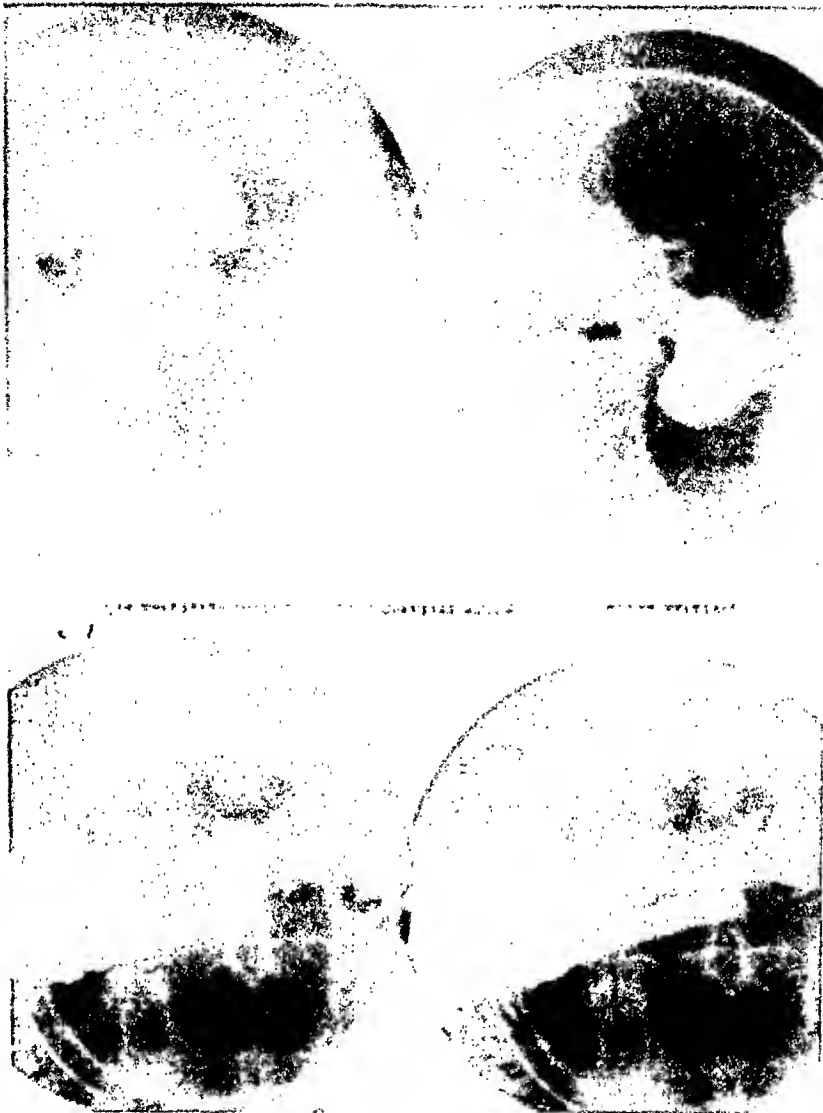
Fig. 7. Follow-up examination of the same patient two years later. Top films disclose the characteristic prolapse of the antral gastric folds into the base of the duodenal bulb. Lower films demonstrate the entirely normal mucosal pattern of the duodenal bulb. The prolapse is not seen in the latter views. The apparent inconstancy of the finding is characteristic of prolapsing lesions.

however, seems within the realm of possibility and may deserve consideration in the evaluation of some acute abdominal conditions. The sudden, profuse gastrointestinal hemorrhage, as observed in our patient, appears the only one of the entire series requiring emergency measures.

Since von Schmieden[2] in 1911 performed a gastrotomy with excision of the hypertrophic mucosal folds and employing a plastic operation to increase the outlet from the stomach, a similar principle has been followed by most of the subsequent operators. In most patients this likewise appears to have given satisfactory results.

It may be debatable whether or not such a procedure will prove entirely satisfactory in individuals with a history of bleeding. In reports of the three patients with associated hemorrhage, recurrence of bleeding followed excision of gastric folds and pyloroplasty.[6,8] With this uncertainty in mind we decided in our patient in favor of a partial gastric resection. Such a proce-

dure was employed in two other subjects in the entire series.[10] In the latter no history of gastrointestinal bleeding was elicited, but the presence of pronounced hyperacidity seemed to warrant the more extensive procedure.

Gastro-enterostomy probably has no place in the surgical treatment of this condition. It has been employed in two instances.[3,6] In one of these[6] a second operation had to be performed with excision of gastric folds and pyloroplasty. In the other patient, co-existence of a duodenal ulcer decided in favor of a gastro-enterostomy and with apparently satisfactory results.

In this connection the varying palpatory findings at the time of operation deserve consideration. In von Schmieden's report a clear description is given of a palpable tumor-like induration in the pyloric area. Similar observations were also made by others,[3,4,7-10] as well as noted in our patient. It seems, therefore, quite probable that the experienced surgeon during a routine exploration in certain patients would be able to put his finger on such an existing gastric pathologic condition even without preoperative positive roentgenologic exploration. It must be recalled, however, that in every one of the operative patients of this series the preoperative x-ray findings indicated the presence of at least abnormal conditions of the pyloric area, if not actual mucosal prolapse. Of greater significance is the statement in several of the reports that careful palpation alone of the antrum and pyloric area gave no confirmatory evidence of the preoperative x-ray findings. Verification of the existing pathologic disease was only obtained after opening into the stomach. In one patient[6] a regular exploratory laparotomy without gastrotomy failed to localize the existing pathologic condition. As the patient's distress continued subsequent x-ray investigations succeeded in demonstration of the mucosal prolapse. Subsequent reoperation with gastrotomy and excision of prolapsed mucosal folds finally resulted in cure.

It may further be of significance to point

FIG. 8. Pyloric ulcer associated with minimal secondary prolapse in another of our patients, a man aged thirty-two. At operation an ulcer the size of a dime was found on the posterior wall of the prepyloric region which had penetrated into the peripancreatic capsule. Partial gastric resection was followed by good therapeutic results.

out the momentary variability of the palpatory findings during operation, as described herein. As a consequence, it will be readily appreciated that a complete and dependable preoperative roentgenologic report in these patients constitutes the essential guide for the surgeon in his decisions regarding problems confronting him during the actual operation.

SUMMARY

1. A review is presented of cases of transpyloric herniation of gastric mucosa. Personally observed patients are included; one of these underwent partial gastric resection because of repeated, severe gastrointestinal hemorrhages resulting from erosion of the extruded gastric mucosa.

2. Future closer attention to this condition will probably prove its presence in a considerably larger number of individuals than is apparent at present. With this in mind it is suggested as probably worth while to review the histories of patients with an indeterminate type of dyspepsia

previously classified as functional disorders. It is anticipated that some of the latter may present positive roentgen evidence of extrusion of redundant gastric mucosa through the pylorus.

3. Transpyloric herniation of the gastric mucosa represents a pathologic entity; it is essentially characterized by an abnormally loose layer of submucosa and redundancy of the antral gastric mucosa. Because of their anatomic position, these redundant mucosal folds are intermittently extruded through the pylorus into the duodenum. In many of the subjects a coexistence of hypertrophic gastritis has been noted. In others, it has been found to be absent. We have personally been impressed by the fact that several of the patients intermittently have been heavy consumers of alcoholic beverages.

4. At least with our present knowledge, the type of dyspepsia present in these patients is of such a diffuse uncharacteristic type as to prevent a definite clinical diagnosis without the aid of the roentgenologist. The very uncharacteristic, sometimes bizarre history, however, may make the clinician suspicious of such a condition and thereby alert the x-ray department to pay particular attention to the prepyloric and pyloric area during x-ray investigation.

5. The possibility of a mucosal erosion of the extruded gastric mucosa must be kept in mind as a differential diagnostic point in evaluation of patients with obscure types of gastrointestinal bleeding.

6. In most patients the roentgenologic picture of transpyloric herniation of the gastric mucosa is a typical one and is represented by a filling defect resembling a cauliflower. It is to be emphasized that, due to peristaltic movements of the individual mucosal folds, the outline of the defect may change its pattern from moment to moment. In order to permit accurate studies, certain technical details must be carefully considered, namely, change in position of the patient, change of views, adequate technic for mucosal studies, avoiding overdistention with contrast media and careful compression technic.

7. In the treatment of this condition it seems logical in most patients to try medical measures first (dietary regime, antispasmodics, correction of abnormal production of gastric juices). Striking improvements are usually not observed, but may be sufficient to allow the patient reasonable comfort and ability to work. In others, the severity of, or rapid progression of the distress particularly when associated with gastrointestinal bleeding, may necessitate surgical intervention.

8. It is of inestimable value to the surgeon preoperatively, to have obtained a correct and complete roentgenologic diagnosis as well as the clinician's careful evaluation of the entire clinical picture. This information represents valuable guidance during the actual exploration and will help him to decide whether or not to perform an exploratory gastrotomy and whether to select as the operative procedure an excision of the redundant folds with pyloroplasty or the more extensive gastric resection.

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A MUSCLE hernia is easily distinguished from varicose veins by the fact that it protrudes when the patient is standing and becomes even larger when he is standing on his toes. It is less prominent when the patient is not on his feet or when he is standing with the foot dorsiflexed, at which time the protrusion may disappear entirely.

From "Surgery of the Ambulatory Patient" by L. Kraeer Fergerson (J. B. Lippincott Company).

REPAIR OF MASSIVE DEFECT OF TIBIA WITHOUT FIXATION*

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A PROCEDURE used by the authors in the treatment of large bony loss of the tibia is offered. This method differs from others in that no metallic or other form of foreign substance is used to maintain position of donor graft. An almost

Our experience has shown us that any fixation of the graft with metals or other foreign materials is not necessary provided the site for reception is well prepared. This opinion is substantiated by the results found in the treatment of fractures by the
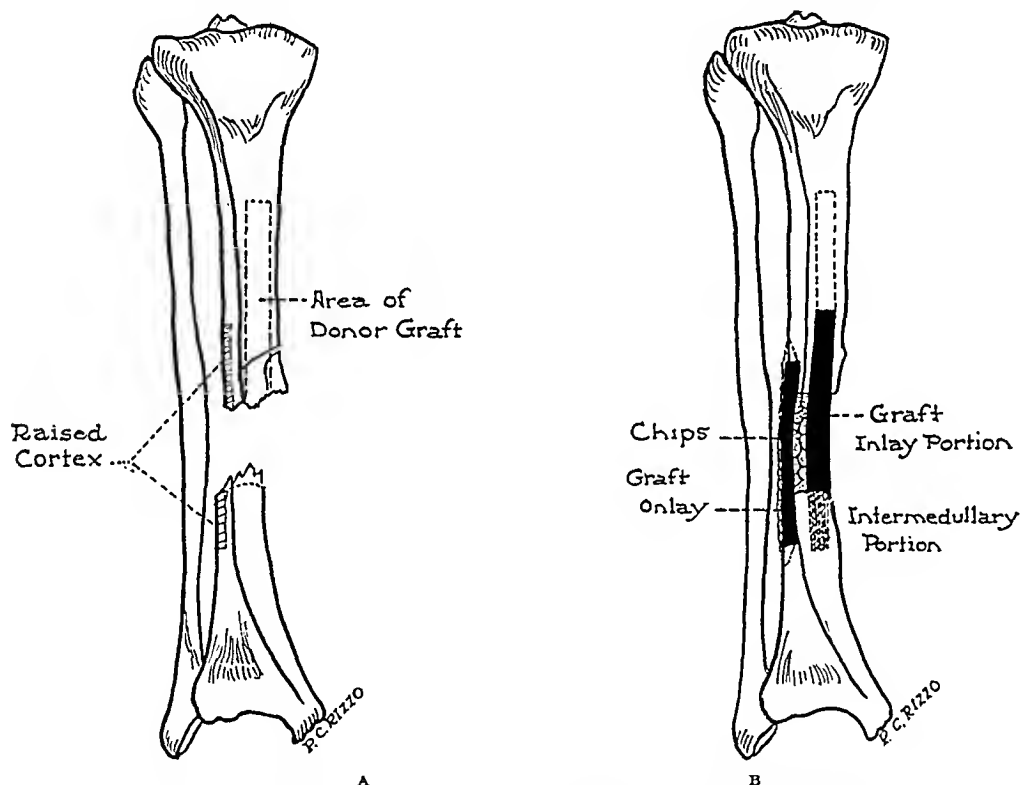

FIG. 1. A, diagram of prepared area; B, diagram of position of grafts.

similar treatment has been used in 200 cases of non-union. The number of cases now being presented are six with 100 per cent success.

We believe that when foreign material is introduced into a healed compound wound the incidences of flare-up and latent infection are imminent. This opinion is gained from experience in patients treated by us with metallic fixation. Of eight patients so treated four needed secondary surgery.

closed method. It can be said with reasonable certainty that most fractures heal, whether comminuted or simple, provided there is good contact of fragments. The greater number of patients are treated by the general practitioner. His experience might be extensive and it might be very meager; nonetheless, in the uncomplicated case he usually gets a good result. Immobilization of these fractures by the general practitioner is accomplished by cast, splints

Fig. 2. Case R. M. A, preoperative x-ray; B, four months postoperatively showing outlines of grafts and healing process.

Fig. 3. Case N. P. A, preoperative x-ray; B, five months postoperatively showing incorporation of grafts and solid healing.

and suspension apparatus. The casts vary from snug skin plasters with excellent immobilization to the thickly and overpadded cast with plenty of room for movement inside the cast as soon as the swelling subsides and the padding becomes compressed.

In some instances splints have been used and most of these are inadequate. Suspension treatment by weights cannot give absolute fixation since the patient is always jiggling about and there is a varying degree of motion at the fracture site. Figures 1A and B describe graphically the procedure we use.

All fibrous tissue is removed from between the fracture ends and as much scar tissue as possible from the surrounding structures. The medullary canal is opened

and all fragments of sclerotic bone removed. Next a donor graft is taken from the longer of the tibial fragments. The longer the graft the more useful it is. The lateral or medial aspect of the tibia is exposed and a thin layer of cortical bone is raised in both fragments. This is to be the recipient surface for one of the donor grafts. Meanwhile a graft has been removed from the opposite leg, usually one measuring 6 to 7 inches by $\frac{5}{8}$ to $\frac{3}{4}$ inch. This graft is inserted into the medullary canal of the shorter fragment and inlaid into the space created by the removal of the donor graft from the larger fragment. The other donor graft, the one removed from the same tibia, is placed in the prepared area on the lateral or medial aspect of the tibia; it is an onlay graft in nature.

Next, bone chips are placed in the space between the two grafts. These chips are usually trimmings from the grafts. The free graft "onlay" is covered and held in place by the leg muscles when the tissue is closed in layers. A long leg plaster is next applied. This is to be changed in six to eight weeks when an unpadded plaster cast is applied. This unpadded plaster cast is used as a walking plaster by attaching a walking iron eight weeks postoperatively. The patient is kept in a walking plaster for four to five months. Check-up monthly x-rays determine the duration of immobilization. A brace is worn for two to four months after this. The duration is again determined by check-up x-ray.

The procedure herein described can be called a combination of onlay, inlay and intramedullary graft. Figures 2 and 3 demonstrate the before and after results in following two cases:

CASE REPORTS

CASE I. R. M. sustained a compound comminuted fracture of the right tibia and fibula on December 3, 1944. Prior to admission to Fletcher General Hospital he had undergone débridement and secondary closure. On arrival his leg was in a long cast. There was complete healing at the site of injury. X-rays showed absence of about 2 inches of the tibia at the middle third. On April 21, 1945, a double bone graft was performed. The cast was removed October 10, 1945. He then wore a brace until December 3, 1945, and was subsequently discharged with firm union.

CASE II. N. P. was wounded in action on November 19, 1944. On admission to this hospital the leg was well healed but x-rays showed loss of about 2 inches of the upper third of the tibia. On April 7, 1945, fusion of the fibula to tibia was done distally and proximally; on June 28, 1945, an osteotomy of the fibula and double bone graft was performed. After five months the cast was removed. Union was so firm that no brace was necessary and he was discharged healed on January 28, 1946.

RISK AND WELL PLANNED SURGERY

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THE question is often asked by laymen, "I wonder if I will be able to stand the operation." Unfortunately, we often find that some medical men treat definite surgical pathologic conditions too long, with the excuse that, "it may save you from an operation." Actually, there are few men in the profession who really believe that such a hazard exists in a major operative procedure that it is perhaps best to treat them with a non-surgical method and that their expectancy of life may be prolonged thereby. Perhaps it was only twenty-five years ago that this was true. In days before that we know it was true.

Today, with all our modern preparations and study, both preoperative and postoperative, with modern anesthesia and technic, nearly any surgical procedure which may be indicated can be carried out successfully regardless of age or condition of the patient.

I believe that any patient with normal heart, lungs and kidneys on which a major surgical procedure is carried out should have 100 per cent recovery, unless some unforeseen accident occurs, if all adequate study and preoperative care, surgical judgment and postoperative care are judiciously employed. We cannot say we have solved surgical problems until we can guarantee such a percentage of recoveries.

We submit a brief record of a fifty-two year old female patient who had nine operations in twenty-five years, eight of them major, by six different surgeons and in all of which definite pathologic disease was shown. In spite of this trouble she has been able to carry out the duties of a normal wife and mother.

CASE REPORT

This patient was first admitted to Mercy Hospital, Columbus, Ohio in September, 1921 on the service of Dr. Joseph Price with the usual history and symptoms of chronic salpingitis. This was long before the days of penicillin, therefore surgical treatment was carried out. A bilateral salpingectomy, left oophorectomy and incidental appendectomy were performed under general anesthesia.

The pathologic diagnosis was chronic salpingitis and hemorrhagic cyst of the ovary. The patient made an excellent recovery from this surgical procedure.

Admission number two was to Mt. Carmel Hospital, Columbus, Ohio, on the service of Dr. Charles Hamilton. This was for severe thyrotoxicosis. The patient's condition was so critical that only bilateral ligation of the thyroid arteries could be performed. After this procedure the patient was discharged and requested to return in a few weeks for thyroidectomy. She stated that she "felt so good" after the operation that she did not desire any further surgery. However, her thyroid condition became so severe soon after a change in her residence that she was compelled to enter the hospital a third time.

This was at Gadsen, Alabama, where a thyroidectomy was performed under local anesthesia in February, 1928, by Dr. Lorage of Gadsen. Again the patient made a good surgical recovery although she must have had a great heart strain as an exophthalmos of some extent still persists.

A fourth admission, and the first one to be entered at the Akron City Hospital, was on November 27, 1931, on the service of Dr. C. E. Jelm with a diagnosis of hydronephrosis of the left kidney, which was confirmed by cystoscopic examination. Surgical operation to free a pelvo-ureteral junction adhesion was performed under spinal anesthesia. Adhesions, which produced constriction of the ureter and the pelvo junctions, were freed. The patient recovered quickly and was freed of her hydronephrosis and of her symptoms.

The patient's fifth surgical admission and the first under the author's service was to the Akron City Hospital on June 1, 1934.

At that time she had the history and physical findings of an acute intestinal obstruction. At an emergency operation under spinal anesthesia we found an acute obstruction caused by about 3 inches of ileum which had become adherent to her remaining and now cystic right ovary. This evidently was a chronic adhesion which had become acute. The ileum had to be dissected from the ovary, multiple fibroid tumors of the uterus were noted but no further surgery was indicated at this time because of the toxic condition of the patient due to intestinal obstruction. Again the patient made a good recovery with the proper postoperative treatment of this chronic and acute complication of intestinal obstruction.

The pathologic findings which we discovered at the time of this admission increased and caused the sixth hospital admission of this patient again at the Akron City Hospital and again under the author's service in March, 1937. On April 1, 1937, we did a supracervical hysterectomy and an oophorectomy on this patient under spinal anesthesia.

The pathologic diagnosis of the material submitted was multiple intramural and subserous fibromyoma of the uterus and large hemorrhagic cyst of the ovary. Once again the patient made a quick recovery from this operation and returned home ten days postoperatively.

Approximately one year later in April, 1938, this patient was again admitted to the same hospital under my service with a history and complaint of service attacks of pain in the left kidney region, the same kidney on which the previous operation of the pelvoureter junction had been performed seven and one-half years previous. X-ray examination showed a large left hydronephrosis with multiple calculi and complete blockage of the ureter. The kidney was non-functioning. On this seventh admission, therefore, we were obliged to perform a left nephrectomy which we did under spinal anesthesia.

The pathologic diagnosis stated left hydronephrosis with marked dilation of the calyces and fibrous atrophy of the cortex. There were five stones up to 3 cm. in diameter. Once again the patient made an excellent surgical recovery and left the hospital thirteen days postoperatively.

While the author was absent during the recent war, this patient had admission and operation number eight. Her complaint at this time was pain in the right upper quadrant and back. She was admitted to the service of an experienced urologist. In a careful review of all these admissions this was the only one about which there may have been a question as to the indication for surgical procedure. A diagnosis of ptosis with one plus hydronephrosis of the right and only remaining kidney was made and right nephropexy under general anesthesia was performed on September 15, 1942.

In my opinion a nephropexy is one operation that should be discarded as a relic of the past. The patient made a good surgical recovery from this procedure and left the hospital on the thirteenth day postoperatively.

Upon my return from military service this patient again came to me with a complaint similar to the one that caused her eighth and last admission. This was in September, 1945. History and physical examinations convinced me that this patient was now and probably had been for some time suffering from gallbladder disease. This was demonstrated by x-ray examinations.

Therefore, the patient was admitted to the hospital for the ninth time. We performed a cholecystectomy, again under spinal anesthesia which the patient tolerated with great ease. The pathologic diagnosis was cholecystitis with lithiasis. The gallbladder contained twenty-four large calculi. Once again this patient made an excellent recovery and was home on the thirteenth postoperative day.

We saw her recently, one year after her last operation, and she appears to be in excellent health with no complaints whatsoever.

CONCLUSION

We have presented the history of a patient on whom nine separate surgical operations by six different surgeons have been performed with definite pathologic disease in each instance. I believe that with careful study, careful diagnosis, good preoperative preparation, accurate surgery, good anesthesia, (spinal preferred) and good postoperative care of the patient, surgery can almost reach the "wonder stage" of relieving pain and extending life.

CHORIONCARCINOMA

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BECAUSE malignant tumors involving fetal parts are infrequently seen during the course of general surgical practice and as their symptomatology is relatively obscure, this type of malignancy is often unsuspected until generalized metastases have occurred.

In 1943, we had the opportunity to treat a patient giving a clinical history of abnormal uterine bleeding of fourteen months' duration. Pathologic examination of the operative specimen revealed an invasive chorioncarcinoma. Statistics indicate that this is an uncommon condition with an incidence of about 0.01 per cent or one in each ten thousand cases of uterine tumors.

Clinical Features and Pathology. Chorioncarcinoma in the female is always associated with pregnancy. It may follow an abortion, more rarely a full term pregnancy and is seen in one-third of patients with a hydatidiform mole. In the male it appears as a testicular tumor probably as the result of embryonic ectodermal inclusions. The interval between gestation and the appearance of the chorioncarcinoma may be as long as three years although generally it is much less. Associated with this tumor is a high incidence of large, bilateral lutein cysts of the ovaries probably a result of rather than a cause for the uterine condition.

For many years the tumor was believed to be of decidual origin because of the similar histologic picture but Marchand established the currently accepted view that it, like the hydatidiform mole, is of fetal origin arising from chorionic villi. The essential unit of the villus is the trophoblast, composed of an outer layer of dark, multinucleated cytoplasmic masses known as syncytial cells, and an inner stratum of large, clear, cubical cells with poorly staining nuclei called Langhans' cells. The purpose of the trophoblast under normal conditions, i.e., pregnancy, is invasion of uterine blood vessels for the formation of an adequate fetal circulation. The malignant activity of chorioncarcinoma which retains trophoblastic characteristics is expressed by it's invasion of venous and arterial sinusoids. Cellular emboli are rapidly and profusely disseminated to distant parts of the body even early in the disease. Usual sites of metastases are the lungs, brain and vagina. The associated poor prognosis is attributable to this metastatic potentiality and the unusually high degree of cellular anaplasia. There is no stroma or blood vessels, the tumor receiving nourishment from the vessels it invades. Microscopic sections show a diffuse hyperplasia of the Langhans' cells with penetration through the syncytial layer into the uterine musculature. It is the Langhans' layer that is the culprit. Cases have been reported of patients harboring chorionepithelioma which exhibited no metastatic tendencies. These were probably examples of syncytiomas or benign trophoblastic tumors composed of predominantly syncytial elements having no invasive characteristics. In this type of tumor the Langhans' cells are conspicuously absent.

Clinically, the tests of pregnancy, depending on the presence of ovarian-stimulating pituitary hormones, are positive in all cases of chorionepithelioma. The Aschheim-Zondek test may therefore be used as an index of existing trophoblastic activity in diagnosis and for determining the completeness of surgical extirpation. Strongly positive Aschheim-Zondek tests are also produced by chorionepithelioma in the male.

Diagnosis. The primary malignancy is usually small and may be unaccompanied by any appreciable uterine enlargement so that eliciting a history of past pregnancy is extremely important. Earlier expulsion of a mole is very significant. Repeated uterine hemorrhage with the appearance of a positive Aschheim-Zondek reaction or increase in the strength of a previously positive test suggest the presence of chorionepithelioma. A *gentle* curettage is indicated and the specimen obtained should be subjected to careful microscopic examination. Curettement of patients with a hydatidiform mole or chorioncarcinoma is not without danger as the thin, weakened uterine wall can be easily perforated even by a dull curet or the finger.

Treatment. In patients with generalized metastases or with parametrial infiltration due to chorioncarcinoma, surgery is useless. Radiation therapy may be circumspectly instituted but the prognosis is uniformly poor. Chorioncarcinoma is among the most malignant of tumors. When the lesions are confined to excisable areas, hysterectomy is the treatment of choice. Postoperative gonadotrophic hormone assays should be repeated to determine the completeness of surgical removal.

CASE HISTORY

Mrs. G. P., No. 322043, was admitted to St. Elizabeth Hospital on September 20, 1943, with a chief complaint of continuous vaginal bleeding of fourteen months' duration. She stated that in 1942, following a normal obstetrical delivery, a slowly, progressive loss of strength and energy developed. She did not feel well and the postpartum lochia remained blood-tinged. Six weeks later, there was a sudden, profuse vaginal hemorrhage accompanied by the passage of large clots, lasting about one hour. Medical treatment, administered elsewhere, consisted of progesterone injections which diminished the bleeding but failed to produce complete cessation. Metrorrhagia continued for one year, varying daily in amount from a slight bloody show to episodes of severe active bleeding necessitating bed rest and sedatives. The patient had lost 25 pounds in weight; there had been no pain or nausea.

Physical examination disclosed a pale, thin female, aged thirty-one years, showing marked air hunger. The head was normal except for slight exophthalmos. The thyroid was palpable and soft with no masses present. Superficial arterial pulsations were prominently visible in the neck, temporal regions, axillae and groin. The lung fields were tympanitic with no adventitious sounds. The area of superficial cardiac dullness seemed slightly enlarged to the left. The apical rate was 102 per minute, regular, with fair valve tones and no pulse deficit or murmurs. The blood pressure was 104/60 and the temperature, 99.6°F. The liver appeared normal in size and slightly ptosed; the right kidney could be palpated. The abdomen was scaphoid and showed evidence of substantial weight loss. There was a large umbilical hernia with diastasis recti probably caused by four past gestations. Abdominal tenderness was absent. Examination of the vaginal introitus disclosed a steady, sanguineous oozing through a clean multiparous cervix. A moderate degree of bladder prolapse and a large rectocele were present. The urethral sphincter tone was fairly good. Bimanual examination of the pelvis revealed a hypertrophied mobile uterus in second procidentia. The broad ligament and adnexa were normal with some suggestion of bilateral ovarian enlargement. In view of these findings, it was considered advisable to eliminate the possibility of fundal carcinoma and a gentle curettage was performed under local anesthesia.

The pathologic report indicated that the "Sections show tissue suggestive of chorioncarcinoma." The Friedman test, with blood serum obtained on the day after curettement, was strongly positive. Visualization of the vaginal mucosa under magnification failed to disclose the presence of malignant implants.

The blood serology revealed the following: white blood cells, 9,000; red blood cells, 3.1 million; hemoglobin, 53 per cent; stabs, 5; segmented, 58; eosinophiles, 1 per cent; lymphocytes, 29 per cent; monocytes, 7 per cent. Urine (catheter specimen), negative; blood group, A and Kahn test, negative.

Diagnosis: Chorioncarcinoma; umbilical hernia; uterine procidentia; cystocele-rectocele and secondary anemia.

On September 22nd, after a blood transfusion of 800 cc., the abdomen was explored through

an infra-umbilical, muscle-splitting incision under pontocaine spinal anesthesia. The uterus appeared grossly normal except for its position which was one of extreme retroversion. The ovaries were both congested, containing multiple follicular and lutein cysts but, in view of the patient's age, it was considered advisable to retain any normally functioning ovarian tissue if possible. The round ligaments were tied with silk and cut on each side, extending the incision from side-to-side through the vesicouterine fold of peritoneum. The bladder was then completely mobilized facilitating careful palpation of the broad ligaments down to the cervix for evidence of possible malignant infiltration. The tissues were soft and flexible. Both uterine arteries were ligated close to their origin and the uterus removed almost in its entirety leaving only a narrow rim of cervical tissue. Suspension of the cervix and vagina was accomplished by fixation to the broad and round ligaments. The bladder was sutured deep behind the cervical stump in an attempt to improve the existing cystocele. Both ovaries were subtotally resected allowing only grossly normal strips of tissue 1 by 3 cm. to remain on each side. These were peritonealized by a Poole suspension. The umbilical hernia was repaired from the peritoneal side by three silk mattress sutures and the abdominal layers approximated in the conventional manner with interrupted No. 40 cotton. The patient was then placed in lithotomy position for repair of the rectocele. It was noted that the intra-abdominal bladder

advance had favorably influenced the cystocele, leaving only a moderate redundancy of anterior vaginal mucous membrane.

The pathologic report revealed the following: The specimen consists of a uterus 60 mm. long by 58 mm. at its widest diameter. This contains a necrotic mass roughly 40 mm. in diameter. It has some gross characteristics of placenta although it seems to be firmly attached to the uterine wall. Sections "show that there are synctial and Langhans' cells which have invaded the uterine musculature. This together with the ovarian cysts makes one think of a true malignancy rather than a burrowing mole. Would suggest running repeated pregnancy tests."

Postoperative convalescence was uneventful. An additional 500 cc. of citrated blood was administered on the day following operation and the Friedman test two days later was negative. A 6 foot chest plate, for possible metastases, was normal. The patient was discharged on September 3rd, the twelfth postoperative day. Three years later, she is in excellent health, two interim pregnancy tests were negative and the patient, we believe, may be considered to have had a complete removal of the chorioncarcinoma.

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TRAUMATIC RUPTURE OF THE THYROID GLAND*

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THIS case is reported because of its rarity.

CASE REPORT

K. K., a male aged forty-six, was admitted to the Saginaw General Hospital at 11 P.M. December 22, 1946. He was the driver of an automobile which skidded on icy pavement and crashed into a telephone pole. Two other occupants of the car were injured, one sustaining comminuted fractures of the femora and some lacerations about the face. The other had comminuted fractures of the right tibia and fibula, contusions of the face and a fracture of the nose. The patient K. K. was found to have a fracture of the right patella and a small abrasion of the neck over the right sterno-mastoid muscle. No evidence of other injuries were noted on admission. There was no shock. The fractured patella was temporarily immobilized by the use of a Cabot posterior splint.

About four hours after admission it was reported that the patient was having difficulty breathing and that there was some swelling of his neck. Ice packs were applied to his neck and a sedative was administered. His breathing gradually became more difficult. He was unable to be comfortable except in a sitting position. Several hours after the onset of orthopnea, the neck was found to be moderately and uniformly swollen, anteriorly. It was firm and tender. While the patient's breathing was difficult, his color was good and his pulse was normal. Examination by an otolaryngologist revealed that the air passages were free. There was no evidence of any compression or displacement of the larynx or trachea. It was believed at that time that there was some hemorrhage under the deep cervical fascia but that conservative treatment was indicated.

Orthopnea increased in severity but at no time was there any cyanosis. About noon on the day following admission, the difficulty in breathing and swelling of the neck had increased to such an extent that it was believed that hemorrhage under the deep cervical fascia was continuing and that surgical intervention was indicated. Swelling of the neck was uniform. All the landmarks were obliterated. The swollen area was of stony hardness to the touch and extremely tender. At 3 P.M. that afternoon an operation was performed. A transverse collar incision was made stripping the skin upward. The skin and platysma were found to be very edematous. The cervical fascia was divided in the midline and a large amount of clot and fluid blood allowed to escape from both sides of the trachea. There appeared to be a complete severance of the thyroid gland at the point at which the isthmus merged into the left lateral lobe. This looked as though it had been cut with a blunt instrument. The isthmus contained apparently some small adenomatous changes in which there were a number of blood vessels of a fairly large caliber. One of these presented an arterial bleed. This was ligated. The torn edges of the gland were approximated loosely with silk. The left side, from which there appeared to be some minor oozing, was packed lightly with iodoform gauze and the wound was closed loosely about this pack with interrupted sutures of silk.

As soon as the blood clot was evacuated, the patient took a long deep breath without difficulty. Convalescence was uneventful. The drain was removed on the second day and the wound healed by first intention.

Ten days later, operative repair of the fractured patella was done. The patient was discharged from the hospital on January 9th and he returned to work as a mechanic on February 18th. At that time he had no residual symptoms whatever suggesting any permanent damage to his thyroid. The range of motion of his knee was normal.

Comment. The character of the injuries of all three patients involved in this accident suggested that they had been violently thrown forward in the car. The exact mechanism of this patient's injury was not determined. He could not recall striking his neck against the steering wheel or any part of the car. At the time of the accident he was wearing a heavy overcoat and a sweater, both of which were pulled well up around his neck. It is presumed that he probably struck the right side of his neck against the steering wheel of the car.

HYPERTHYROIDISM results from an excessive absorption of the thyroid secretion (*thyroxin*) and is characterized by the stimulation of nearly every bodily function. A toxic goiter is one characterized by hyperthyroidism, or in the case of a diffuse toxic goiter with exophthalmos, possibly a toxic dysthyroidism.

From "Principles and Practice of Surgery" by W. Wayne Babcock (Lea & Febiger).

New Instrument

NEW TYPE APPARATUS FOR GIVING INTRAVENOUS ANESTHESIA

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SEVERAL intravenous apparatuses have been described in the literature but none have offered the completely closed system with the compactness, ease of operation and control as the one herein described.

While the author was acting as anesthestist during the war, he developed the apparatus herein described. It has been used over a period of three years and has been found very satisfactory. Pentothal sodium was the drug used in every instance.

The apparatus* as shown in Figure 1 is essentially an instrument of simple construction which is a very great aid in administering intravenous pentothal sodium anesthesia.

The body (R) is made of cast aluminum and clamped flat on the arm board by means of a bracket (S) which is also constructed to hold the reservoir bottle (F). It is distinctive in its simplicity and compactness, being so small that it does not project up into the way of any of the operating equipment. Furthermore, the feeder screw (A) which operates through the sliding plunger head holder (D) gives more accurate control of the syringe plunger (E) with less friction. The rotary type syringe barrel clamps (J) are efficient and easily operated.

The bracket which clamps the apparatus to the table is so constructed that it may easily be transferred to the opposite side

* The apparatus is manufactured and distributed by the Kansas City Assemblage Company, Kansas City, Mo.

by merely loosening a thumb screw (Q). Since the bracket also holds the reservoir container the latter may be put on the side where it will be most convenient.

The accessory equipment consists of a small wide mouthed bottle (F) 2 inches in diameter and 2½ to 3 inches high, two 8-inch pieces of ⅛-inch inside bore rubber tubing (L), one three-way valve (K), a rubber stopper (G) containing two holes, one small glass observation needle adapter (N), one 20 cc. Becton, Dickinson syringe (I), a 20-gauge 1½-inch hypodermic needle (M) and two pieces of glass tubing connected with the stopper.

A stand is provided so that the apparatus may be assembled, the syringe, tubing and bottle filled with pentothal solution and all set aside conveniently until the anesthetist is ready to start anesthesia.

When the entire apparatus is set up as shown in Figure 1 and connected with the vein in the arm, it occupies so little space that sterile operating sheets may be thrown over it all. Operation of the equipment beneath the sheets is very easy and a measure of safety to both surgeon and anesthetist is added.

TECHNIC

The tubing with three-way valve and glass adapter, two 20 cc. syringes, two drug glasses, two 20-gauge 1½-inch needles, the reservoir bottle (with the rubber stopper) and some gauze sponges are wrapped and sterilized in an individual kit. The syringe plungers are coated with a little glycerine
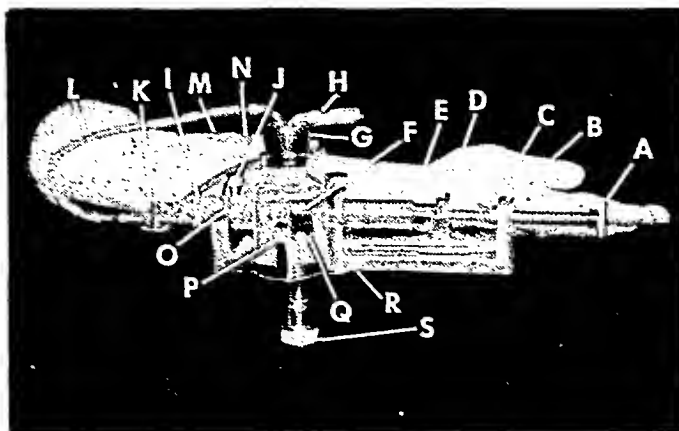
FIG. 1. A, feeder screw; B, set screw; C, I. D. threaded bushing; D, syringe plunger head holder; E, syringe plunger; F, reservoir bottle; G, rubber stopper; H, air vent tube; I, syringe barrel; J, rotary syringe clamp; K, three-way valve; L, rubber tube from syringe to supply bottle; M, hypodermic needle; N, glass adapter; O, holder for needle and tube when not in use. (The other cut opposite side of apparatus.) P, sliding arm of bracket. (The other cut opposite side of apparatus.) Q, thumb set screw to hold bracket in place. (The other cut opposite side of apparatus.) R, main body of holder; S, bracket for holding bottle and also for clamping apparatus to table.

before sterilizing. The syringe should be tested in the holder to be sure it fits before sterilizing.

When it is desired to give an intravenous anesthetic, the apparatus is assembled after checking all parts carefully. It is especially important to manipulate the three-way valve several times to be sure it works easily. Otherwise, the shank of the syringe may be broken off in the process. The Becton, Dickinson Luer-lok type syringe is safer to use and is included in the set.

Next, the pentothal powder is put into the medicine glass and sufficient sterilized distilled water (40 cc. per Gm.) added to make a 2½ per cent solution. When completely dissolved, the syringe and tubing are filled with solution, being sure to eliminate all air bubbles. The excess solution is put into the reservoir bottle and the stopper snugly applied. The reservoir bottle is fitted with a No. 6 stopper containing two holes. In one hole a glass tube connects the rubber tubing and syringe to the bottom of the jar for delivering the reserve supply of anesthetic solution. Into the other hole of the stopper a glass tube is placed to act as an air vent (H). In the

latter tube is an enlarged chamber into which is placed some cotton to filter the air going to and from the jar.

When the unit has been assembled and filled with anesthetic solution, the syringe and bottle are fitted into the apparatus for clamping on the table. An alcohol sponge may be put on the needle before putting it into the clamp (O) on the apparatus. The entire apparatus is now set aside on the stand until the anesthetist is ready to start anesthesia.

When ready to start the anesthesia, the anesthetist clamps the apparatus on the armboard beside the patient's arm; the needle is inserted through the skin and the feeder screw reversed one-fourth turn to produce a back pressure so that the blood will immediately appear in the glass adapter as the needle punctures the vein.

The latter procedure will obviate the use of a syringe until the vein is found as is necessary in some obese patients. In fact, this works better than a syringe because the back pressure is maintained until the vein is punctured. This is a great advantage over other methods.

The anesthetic material is now injected slowly into the vein until the desired amount is given. A slight turn of the feeder screw may be made at intervals, if necessary, to keep blood from coagulating in the needle. When the patient is at the desired depth of anesthesia, the anesthetist's hands will be free to do other things. It is a great advantage to the anesthetist to have perfect control of the anesthesia and at the same time to be free to work with the patient at all times.

When the syringe is empty, one may change the three-way valve and reverse the feeder screw to refill from the jar. The valve is again changed and the anesthetic solution forced into the vein by a few turns of the feeder screw as needed. If the plunger seems to stick, give it a turn with the fingers and smooth operation will follow. However, "jiggling" the feeder screw back and forth slightly may be all that is needed.

The reservoir will hold enough solution for two or three patients and by merely changing the needle and tubing that delivers the pentothal from the syringe to the vein, one may give several anesthetics with perfect safety. Additional pentothal solution may be added to the reservoir bottle whenever desired.

If, at times, one does not wish to use the feeder screw, it may be left in the extreme distal position and the syringe plunger manipulated with the fingers. The apparatus still will be found useful to hold the syringe and its supply of pentothal solution.

Since some things made of glass vary considerably in their dimensions, it is sometimes helpful to turn the syringe in the apparatus to find the position that will give the smoothest operation. The apparatus is made to take a standard Becton, Dickinson 20 cc. syringe although others may be used.

It is believed that if one will familiarize himself with the mechanisms and use the apparatus a few times, he will not care to give intravenous pentothal without it.

SUMMARY

1. A new type apparatus for administering intravenous anesthesia is described.

2. The apparatus is compact, easy to operate and provides a safe means of giving intravenous pentothal sodium.

3. The administration of the anesthetic is completely under control at all times.

Editorial

RECENT DEVELOPMENTS IN TREATMENT OF HYPERTHY-ROIDISM

DISCOVERY of thiouracil and, more recently, of propylthiouracil has reawakened interest in the possible non-surgical care of hyperthyroidism.

It became apparent at an early stage that thiouracil was not only a dangerous drug but that in the great majority of instances it failed to effect a permanent cure in this disease. Here and there an occasional successful result was reported, usually in an incipient case or in a persistent or recurrent mild case of exophthalmic goiter. The high incidence of complications and, in particular, of the often fatal agranulocytosis induced by its use soon caused most physicians interested in thyroid disease to largely abandon its use and rely upon the traditional method of thyroidectomy. There remain certain definite indications for its use despite its hazards. Until this drug was discovered no effective method of preparing the majority of patients with toxic adenomas for operation existed, iodine being only slightly beneficial in one-third of these subjects. Thiouracil so improved many of these bad risk patients that surgery could be performed with comparative little risk. Otherwise, thiouracil was not indicated in the treatment of hyperthyroidism with two possible exceptions, namely, to give a rest to iodine-fast patients with exophthalmic

goiter and to tide over term the rare case of this disease occurring in late pregnancy. Even here some question exists as to the possible harmful effect of the drug upon the fetus. The concensus of opinion of most workers in the thyroid field was that patients should not be subjected to weeks and months of treatment with a dangerous drug when their health could quickly be restored by the long-accepted method of thyroidectomy.

A new drug, propylthiouracil, was then made available for research study. Investigation in over 2,000 cases in the past two years has definitely shown the merits of this antithyroid drug in contrast to thiouracil. As a consequence of this clinical study the drug was made available to the medical profession in July, 1947 and is now in general use.

Only a few papers and reports have been published on the results of the investigation of this new drug. While it is far less toxic than its predecessor, Bartels has reported one case of agranulocytosis from its use. In a series of 150 patients in whom the drug has been used no such case has occurred in the experience of the writer although the white blood count did fall to 2,500 in one instance. A few cases of skin reaction, vertigo, nausea and vomiting have been reported but, on the whole, far

fewer reactions have occurred than with thiouracil.

What of its effectiveness? Briefly it may be said that, like thiouracil, it will control but not cure hyperthyroidism. In a series of thirty-five cases recently studied, in which the patients had received the drug for periods of six to twelve months, in every instance but one, hyperthyroidism recurred within two months. There may be an occasional case, either mild or incipient, of exophthalmic goiter in which this drug will effect a cure, but a much longer period of study will be required to prove this.

What are the indications for its use? Since it is far less toxic and a great deal safer, it should entirely replace the more dangerous thiouracil. Undoubtedly, it was a mistake to have released the latter drug before a longer period of investigation had been undertaken. The large number of deaths reported and unreported that have resulted from the use of thiouracil has probably outweighed the good it has accomplished in the preoperative treatment of advanced cases of toxic adenoma.

It is in this latter group of cases that propylthiouracil has proved its greatest value. In these patients who so often fail to respond to Lugol's solution, digitalis or any other method of therapy, metabolism may be returned to normal and fibrillation and decompensation controlled. After weeks or months of this preparation the patient is conditioned for thyroidectomy which may be performed by an experienced thyroid surgeon, with little more risk than in the non-toxic group. It should be remembered, however, that iodine should be given for at least three weeks before operation and that it is desirable to stop propylthiouracil a week before this time. Otherwise, the greatly increased vascularity of the gland will cause considerable technical difficulty with the thyroidectomy and will necessitate the use of thrombin, gelfoam, koagamin and other coagulation substances.

There seems little indication for using this drug in the average patient with exophthalmic goiter who might be better prepared in the traditional way with Lugol's solution for ten days before operation. In the exceptionally bad risk patient, in the aged, in children, in the iodine-fast or in the pregnant patient it may be advisable to try the drug as a means of controlling the disease for the present time. Further study may show that longer periods of treatment, years rather than months, may effect a cure in some of these cases. However, there is no proof as yet that such prolonged periods of treatment may not result in permanent damage to certain organs such as the liver, spleen or bone marrow.

One of the problems introduced by this drug, as is the case with Lugol's solution, is that of increased difficulty in diagnosing a possible case of hyperthyroidism. When a young patient with a borderline case of hyperthyroidism has been given iodine for three weeks, the absence of all signs and symptoms of exophthalmic goiter may fairly well mask the picture and confuse the most experienced diagnostician. Propylthiouracil does the same thing when it is used over a long period of time; it is often prescribed by physicians not familiar with the disease and it may be expected to considerably confuse the problem of diagnosis. Undoubtedly the majority of physicians realize the importance of first making a careful examination and of obtaining an accurate history and a metabolism test before administering any drug to a patient with suspected hyperthyroidism. The problem then becomes simpler from the diagnostic aspect.

Most surgeons interested in the thyroid field now agree that an adenomatous goiter should be removed when it is ascertained to be present unless there are special contraindications to surgery. Adenomatous goiters, like gallstones, should be removed while they are still potential sources of fatal or serious complications. When hypertension, permanent myocardial damage or cancer has developed, it is not the time to perform thyroidectomy.

Thiouracil and propylthiouracil have

proved valuable aids in the treatment of hyperthyroidism. They will control but not cure hyperthyroidism. They have supplemented but not supplanted thyroidectomy.

No attempt has been made here to evaluate radioactive iodine because it is still in the clinical investigative stage. At the last meeting of the American Association for the Study of Goiter radioactive iodine was shown to destroy certain areas of cancerous goiters but to have no effect on other parts of the gland. In exophthalmic goiter, it may abate the hyperthyroidism, but as yet sufficient time has not elapsed to determine what will be the end results. Whether or not patients thus treated will develop myxedema in later years remains to be determined.

ARNOLD S. JACKSON, M.D.
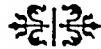
Original Articles

ADENOMA OF THE BRONCHUS*

REVIEW OF FIFTEEN CASES

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MEDICAL literature concerning bronchial adenoma appeared sparingly since Mueller's first description of this disease which he discovered at autopsy and reported in 1882. However, during the past decade an increasing number of reports have appeared. Today bronchial adenoma is widely recognized as a definite clinical and pathologic entity. This tumor, which accounts for approximately 80 per cent of the benign bronchogenic growths, is still a subject of much controversy and as yet no unanimity of opinion exists regarding its histologic origin, potential malignancy, relationship to cancer of the bronchus and its proper treatment.

It is the purpose of this paper to discuss the clinical manifestations, pathologic patterns, available methods of diagnosis and treatment, and to present an analysis of fifteen proven cases of bronchial adenoma studied by the authors, all of which were treated by surgical extirpation, ten by pneumonectomy and five by lobectomy.

GENERAL CONSIDERATIONS

Evidence has accumulated in considering these tumors potentially invasive and malignant. In recent articles [1-5] it has been definitely shown that the so-called benign adenoma does metastasize locally and occasionally to distant organs. Not infrequently surgical specimens demonstrate invasion of adjacent tissue, infiltration of the bronchial wall and metastasis to tracheobronchial lymph nodes.

If one follows the criteria set forth by Graham and Womach [6] for carcinoma which are: invasion of the adjacent tissue, involvement of regional lymph glands and metastasis to distant organs, it can be concluded that in a few cases this entity, although benign, is an incontrovertible evidence that at least some of these tumors have fulfilled all of the requirements of malignancy. However, many still regard with skepticism the possibility of a malignant transformation of these growths. Jackson and Konzelman [7] state that they do not believe that adenoma has an inherent tendency to metastasize or in any other way become malignant. Although the course of the disease points toward benignity, we believe that it can be accepted as an entity which has definite malignant characteristics. This is borne out by the fact that bronchial adenoma not infrequently shows local invasion, involvement of the bronchial wall and occasionally metastasis to the regional lymph nodes. Aside from potential malignancy the tumor eventually produces sequelae incident to bronchial obstruction, namely, obstructive emphysema, total atelectasis, chronic pneumonitis, bronchiectasis, pulmonary abscess and empyema.

In our series of cases one was reported by the pathologist* in 1933 as cancer unclassified, but later was changed to infiltrating adenoma. This patient had a right pneu-

* Dr. Shields Warren, New England Deaconess Hospital, Boston, Mass.

monectomy. Another case was reported as carcinoma of low grade malignancy, suggesting relationship to bronchial adenoma, with metastasis to one regional lymph node. This patient was operated upon seven years ago by right pneumonectomy.

We believe that a comparative study between bronchial adenoma and carcinoma groups will clearly show that these conditions rather than belonging to two grades or stages of the same neoplasm should be considered as being different. The salient

TABLE I

SALIENT FEATURES OF BRONCHIAL ADENOMA AND BRONCHOGENIC CARCINOMA

	Bronchial Adenoma	Bronchogenic Carcinoma
Average age	37.4 years	53.2 years
Sex	Male to Female 1:1	Male to Female 3.8:1
Average duration of disease before surgery is instituted	5.9 years	11.3 months
Clinical picture	Hemorrhages, often profuse, with sudden onset and termination; intermittent episodes of pneumonitis with healthy intervals; bronchiectasis common	Hemorrhages usually blood streaked and continuous; progressive secondary pulmonary complaints without healthy intervals
X-ray findings	Tumor frequently not visible; only shadows incident to obstruction are seen	Tumor frequently is seen
Bronchoscopic picture	Often pedunculated, small, pink, or yellowish in color; carina always sharp and mediastinum free; biopsy frequently followed by bleeding	Usually irregular, fungating, ulcero-granulomatous, non-pedunculated, gray or grayish-yellow; carina frequently, blunted and mediastinum fixed; bleeding minimal
Metastases	Occasionally regional lymph nodes; rarely distant	Regional and distant metastases frequent
Amenability to surgical cure	Almost 100%	10%
Survival	Long duration	Short duration

Another patient who had a left pneumonectomy five years ago demonstrated infiltrating adenoma of bronchial origin (malignant). Two later patients were both operated upon four and three years ago, respectively, both showing infiltrating adenoma of the bronchus. Another patient, operated upon one year ago, demonstrated during operation infiltration of the bronchial wall and the presence of metastases to two lymph nodes. All of these patients are now enjoying good health.

Striking differences in age and sex groups between bronchial adenoma and carcinoma strongly suggest that they are fundamentally different. Alexander,[8] in a recent discussion of bronchial adenoma, stated that their pathologist, Weller, has for many years insisted that the so-called adenoma is a Grade I carcinoma.

differences between these two entities are shown in Table I.

CLINICAL PICTURE

The slow-growing tumor which inevitably but gradually occludes the bronchus will present a definite clinical picture depending upon its location, size and vascularity.

In the early stages the disease is usually asymptomatic but not infrequently, however, a dry, irritating cough is present. Hemoptysis is a cardinal symptom, is often profuse and is as sudden in its termination as in its onset. In women the attacks may coincide with the menstrual periods. Later, as the tumor encroaches upon the lumen of the bronchus, symptoms of partial or total obstruction may appear. A localized wheeze may be noticed by the patient. Dyspnea is

another frequent symptom present at this stage. Usually there have been episodes of pneumonia characterized by cough, mucopurulent sputum, fever and chest pain. These attacks of pulmonary infections are usually severe and slow to resolve.

As the tumor progresses, filling the lumen of the bronchus, obstructive effects with irreparable damage to the lung develop, as in bronchial carcinoma, so that the clinical picture is then one of either atelectasis, bronchiectasis, lung abscess or empyema.

DIAGNOSTIC PROCEDURES

Although the clinical history in most instances is suggestive, a plain roentgenogram will present further evidence especially related to the secondary effects caused by obstruction. The tumor itself is demonstrated only occasionally because of the inflammatory changes overlying it. However, the use of body section roentgenography will permit visualization of the tumor itself as a sharply defined round mass.

Bronchography[9] also allows reasonable accuracy in the delineation of the typical cap-shaped defect in the bronchus. The bronchogram is also informative as to the degree of damage caused distal to the lesion.

Bronchoscopy is still the most decisive single diagnostic procedure available. These tumors almost invariably arise in the major bronchi and are easily accessible for biopsy and histoligic study. The bronchoscopic appearance is typical. Usually a mobile, polypoidal, soft and smooth, pinkish, rounded mass can be seen protruding into the bronchial lumen. These tumors are pedunculated and attached to the bronchus by either a narrow or broad base, occasionally being sessile. They are covered with bronchial mucosa and sometimes are traversed by vessels. Ulceration is rarely seen. The intramural type, with a broad induration, can be mistaken for carcinoma. Histologic verification of adenoma is not always possible because the biopsy specimen may not contain tumor cells but only overlying epithelium and stroma. In other instances biopsy is not advisable because of the possibility of severe and fatal hemorrhage. Occasionally the lack of differentiation of the cells leads to an erroneous diagnosis of carcinoma.

Exploratory thoracotomy we believe should never be delayed when previous methods have failed to substantiate the diagnosis of adenoma.

PATHOGENESIS

The discrepancies regarding the histologic origin of bronchial adenoma accounts largely for the diversity of nomenclature. Several theories of origin have been suggested.[4] Fried expressed the belief that they arise from bronchiomucous glands. Reisner and Wessler and Rabin expressed the opinion that they arise from the duct epithelium of bronchial mucous glands. Goldman and Stevens suggested that the epithelium of either the ducts or the glands may be the site of origin. Mallory states that this tumor resembles carcinoids of the intestinal tract. According to Womack and Graham, the resemblance of adenoma to fetal lung is so striking that it would be extremely difficult, if not impossible, to distinguish one from the other on the basis of microscopic section. They also designate this condition as mixed tumors of the lung because of their observation of similar characteristics seen in mixed tumors of salivary glands. According to them, these tumors contain both endodermal and mesodermal elements. They further state that other anomalies of various kinds are not infrequently associated. They have found during operation either absence or excessive lobulations and congenital cystic disease. Jackson and Konzelman[7] indicated that they were not convinced of an origin from the epithelium of the mucous glands but merely state that the bronchial adenoma shows a peculiar epithelial proliferation in an equally peculiar stroma.
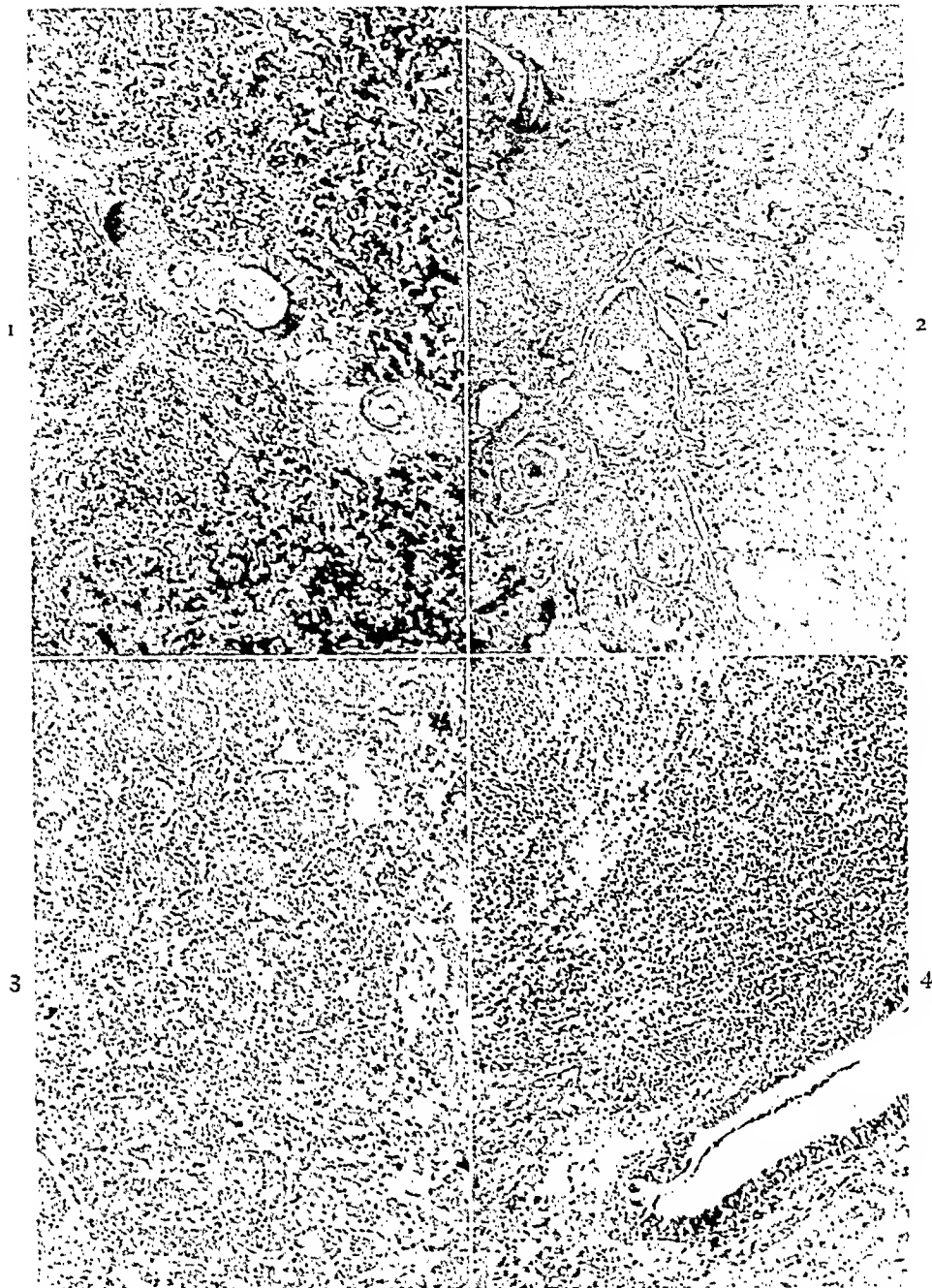
Histologically, bronchial adenoma is

Fig. 1. Case x demonstrates bronchial adenoma showing infiltration between bronchial mucosal glands.

Fig. 2. Case xi demonstrates bronchial adenoma. Tumor cells relatively large with vesicular nuclei and clear cytoplasm; some attempt at acinus formation.

Fig. 3. Case vi demonstrates infiltrating adenoma. Note cord-like pattern of cell clusters; occasional small acinar lumen formed.

Fig. 4. Case ix demonstrates infiltrating adenoma which had metastases in two lung hilar lymph nodes. Here the tumor is projecting into the bronchial lumen. Type cell relatively small with hyperchromatic nucleus; nuclei often eccentrically placed, resembling plasma cell. There is some attempt of the cells at alignment into cords.

characterized by the rarity of mitotic figures, tendency for the cells to be grouped and uniformity of cell type. It is marked by variability of form. The cells are small, cuboidal, polygonal and contain a dark nucleus. They are grouped in a variety of patterns which may be alveolar, columnar, nodullary or mosaic in type depending upon the arrangement of the stroma which divides the cells into groups.
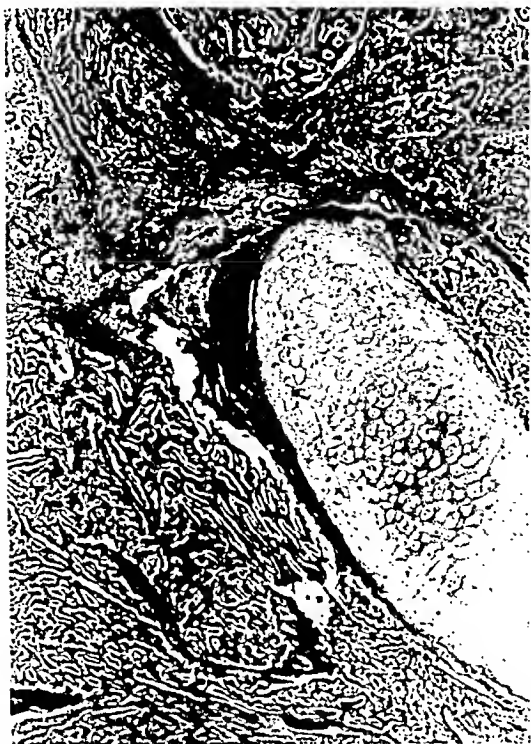
FIG. 5. Case viii shows the more common histologic picture, suggesting a carcinoid type. Note infiltration around cartilage.

The variable histologic pictures of five representative cases in our series are shown in Figures 1 to 5.

TREATMENT

The methods of treatment of bronchial adenoma are three: (1) Radiation therapy; (2) bronchoscopic treatment including (a) implantation of radon seeds, (b) electrocoagulation and (c) forceps removal; (3) surgical extirpation consisting of (a) lobectomy and/or (b) pneumonectomy.

Radiation therapy as a form of treatment has little effect upon the tumor and is not without danger. Foster—Carter has found irradiation efficacious but reports a few deaths from hemorrhages after treatment, apparently as a result of necrosis of the bronchial wall and erosion of blood vessels.

Case v in our series was treated by radiation therapy, receiving 1,200 R units in thirty applications over a period of thirty days. Later a bronchoscopy still revealed the presence of the tumor. Treatments by bronchoscopic implantation of radon seed and electrocoagulation were immediately instituted. While it is true that no evidence of the tumor was found during the operation, it is interesting to note that the lung specimen revealed extensive fibrosis and bronchiectasis necessitating surgical extirpation. Obviously this was a radiation therapy failure. Bronchoscopic treatment of benign endobronchial tumors by implantation of radon seeds, electrocoagulation and forceps removal are methods practiced mainly by most bronchoscopists. We, however, firmly believe that these procedures carry with them numerous important disadvantages, namely, (1) Local recurrence after removal; (2) technical difficulties associated with removal because of size and broad implantation; (3) it is incomplete because of the intra- and extra-bronchial extension of the tumor as shown in Case xii; (4) danger of fatal hemorrhage; (5) persistence of the irreversible suppurative changes distal to the growth (Case v) and (6) presence of regional metastases.

Implantation of radon seeds were used in Case v supplementing radiation therapy without success.

Electrocoagulation, according to Jackson, seems to be a safer procedure than the piecemeal forceps removal since it has the advantage of minimizing the chances of bleeding.

Bronchoscopic removal should be utilized only in patients who refuse surgery, or as a preoperative attempt to bring about drainage and temporary improvement in chronic pneumonitis.

Inasmuch as the use of bronchoscopic treatment for removal of bronchial adenoma is therefore limited and in view of the fact that bronchial adenoma has been proved to recur locally and to be potentially malignant, we believe that surgical removal either by lobectomy or pneumonectomy should be accepted as the primary treatment of choice. The extra-bronchial extension of the tumor and the added irreparable pulmonary damage makes surgery the only rational form of therapy.

We have reviewed all of the bronchial adenoma and carcinoma cases treated by Overholt since 1933. Three hundred five histologically verified cases of pulmonary carcinoma and fifteen of bronchial adenoma were included in this study. Bronchial adenoma constitutes 3.6 per cent of the patients considered. The average age for carcinoma was 53.2 years and for bronchial adenoma 37.4 years when surgery was instituted. It is uniformly accepted that the latter disease occurs more frequently in women and the former in men. In our series the ratio between male and female was, in the cancer group, 3.8 to 1 and in the adenoma group 1 to 1.

Although adenoma of the bronchus, as a group, compared to carcinoma appears small, it assumes a great importance when one considers that they nearly all are operable and curable. In the 305 cases of the pulmonary carcinoma group eighty-seven were thought to be suitable for resection. Of these, thirty-seven had a palliative pneumonectomy because of metastases. Of the carcinoma group, fifty had a curative resection compared to the twelve of the adenoma group. Therefore, among the curable bronchial neoplasms, adenoma comprises about one-fourth of the group.

CASE REPORTS

CASE I.   T. A., a female aged fourty-six, was admitted to the hospital October 24, 1944. The patient was first taken ill in 1933, at which time she complained of cough, bloody sputum, chills and fever. X-ray showed a round, well circumscribed tumor mass about the size of a tangerine in the right lung near the hilus. Bronchoscopy revealed a smooth growth obstructing the lower lobe bronchus. The tumor was vascular and bled easily. Bronchoscopic biopsy was negative. She had a right pneumonectomy on November 13, 1937 with a suspected diagnosis of carcinoma. Gross specimen revealed a moderately firm, slightly resilient, definitely circumscribed, tumor mass, 4 cm. in diameter, at the hilus of the middle lobe. The tumor tissue filled one of the main bronchi and compressed the surrounding lung tissue. The bulk of the tumor distended the bronchus to the middle

and lower lobes with greater involvement of the middle lobe. There was no evidence of invasion except at the posteromedial aspect of the tumor where it merged with the bronchial wall. It was readily shelled out from the surrounding lung tissue. Many enlarged lymph nodes were found to be negative.

Comment. This case demonstrates that it is not always possible to remove representative tissue for biopsy because of the capsule surrounding the tumor and the thickened mucous membrane overlying it. It also proves that the diagnosis of these tumors is often a histologic puzzle. This tumor was first diagnosed as carcinoma unclassified. However, a later review of the slide by Dr. Shields Warren was regarded as consistent with an infiltrating bronchial adenoma. At the present time, twelve years after operation, the patient is enjoying excellent health which proves that this type of tumor is benign.

CASE II.   C. M., a female aged thirty-seven, was admitted to the hospital February 28, 1938. Eight years prior to admission she had two episodes of hemoptysis, raising over a cupful of fresh blood, following grippe. During the following two years she had frequent, dry, coughing spells. Two years later x-ray of the chest revealed a thickened pleura. Between 1932 and 1936 she had no hemorrhages. In 1936 she had a severe hemorrhage, following which she had hemoptyses coinciding with her menstrual periods. Occasional episodes of dyspnea occurred. Physical examination was non-contributory. X-ray revealed atelectasis and cystic disease of the left lung with displacement of the mediastinum to the left. Bronchoscopy revealed a protruding mass about the level of the left upper lobe bronchus. This mass was soft, pinkish in color, with a wide base and bled easily. Biopsy was reported as adenoma, benign, epithelial tumor of the bronchus. Left pneumonectomy was performed on March 11, 1938. The lung was atelectatic and a tumor mass was palpated in the upper lobe. The gross specimen removed showed a well circumscribed, coarsely nodular, elastic, pinkish-gray tumor mass firmly attached to the posterior bronchial wall at the level of the upper lobe orifice and measuring 5 by $3\frac{1}{2}$ by 2 cm. Distally, bronchiectasis and

cystic formation were present. Histologic diagnosis was compatible with the so-called bronchial adenoma.

Comment. Although a bronchoscopic biopsy reported this case to be a benign bronchial adenoma, it was obviously not suitable for bronchoscopic removal because of the following: the danger of profuse hemorrhage, extra-pulmonary invasion and irreparable pulmonary damage. In spite of the duration of the disease no metastatic lymph nodes were found, indicating that regional mestastases do not always occur.

CASE III. W. M., a female aged eighteen, complained of hemoptysis and cough of one and one-half years' duration. She was admitted to the hospital on December 5, 1938. One year prior to admission the patient had hemoptysis of a teaspoonful of bright red blood. Five months later she developed a dry, hacking cough. Four months later she again had a severe attack of hemoptysis and was referred to a tuberculosis sanatorium for study. A diagnosis of tuberculosis was ruled out. X-rays on February 3, 1938, showed increased markings in the right middle and lower lobes with apparently a slight decrease in volume of these areas and a moderate shift of the heart and mediastinum toward the right. There was an oval, homogeneous opacity, measuring approximately 2 cm. in diameter, at the level of the seventh interspace extending outward from the right hilum. Radiological diagnosis: "Partial atelectasis, right middle and lower lobes, most likely on the basis of an obstructive lesion near the right hilum. The appearance is strongly suggestive of a new growth." Lipiodol instillation showed a persistent, round shelf lateral to the lower part of the right hilum about 1½ cm. in diameter. There was also a defect in the filling of the lower lobe bronchus although some of the oil passed into one bronchus which was bronchiectatic. Both the middle and lower lobes failed to fill with lipiodol. The impression formed was that there was a bronchial tumor present causing occlusion of the right middle and lower lobe bronchi and a questionable adenoma. Bronchoscopy on December 6, 1938 was reported as follows: "In the right main stem bronchus a tumor mass was seen which practically completely filled the bronchus except for some air passage laterally. This tumor was dark in color,

somewhat pedunculated, although firm on pressure, and bled easily with manipulation. Further inspection of the mass showed it to arise, primarily, from the anterior wall of the right main stem bronchus approximately 1½ cm. below the level of the orifice of the right upper lobe bronchus. Diagnosis: Benign adenoma." On January 29, 1939 a right pneumonectomy was performed. A gross specimen revealed two, firm nodules in the middle lobe, one posterolaterally, roughly 2 cm. in diameter which on cut section was dark, red-brown in color and finely granular. The largest one, near the hilum, measured 5 by 3 by 2 cm. Cut surface showed a circumscribed, moderately soft nodule of pinkish-gray tissue. The middle lobe bronchus was opened and projecting from the wall was seen a polypoidal mass, moderately firm, measuring 3 by 1 by 0.8 cm. in diameter. The bronchus distal to this was plugged with mucoid material. A gland measuring 2.5 by 1½ cm., soft and pinkish-gray, was also removed. The microscopic diagnosis of all of these removed specimens was reported, "Carcinoma of low-grade malignancy, suggesting relationship to bronchial adenoma."

Comment. Because of her age and hemoptysis, a diagnosis of tuberculosis was made. Bronchoscopy, however, revealed the proper diagnosis. Bronchoscopic removal was not attempted because of the presence of an intra-pulmonary mass which was visible on x-ray. In spite of the short duration of the disease, metastatic glands were found in the pulmonary hilum. A bilobectomy, removing the middle and lower lobes, would have been sufficient to eradicate the disease, but because of the metastatic growth in the lymph node pneumonectomy was elected.

CASE IV. I. S., a male aged forty-eight, was admitted to the hospital on October 28, 1940. Four years prior to admission he developed an acute pneumonic process on the left side with temperature, productive cough and pleural pain. The following four years the patient had several episodes of pneumonia. X-rays at that time revealed a tumor mass in the left hilum. Patient ran a septic course for three months with high temperature and a large amount of mucopurulent sputum. Bronchoscopy revealed a soft, glistening, reddish-
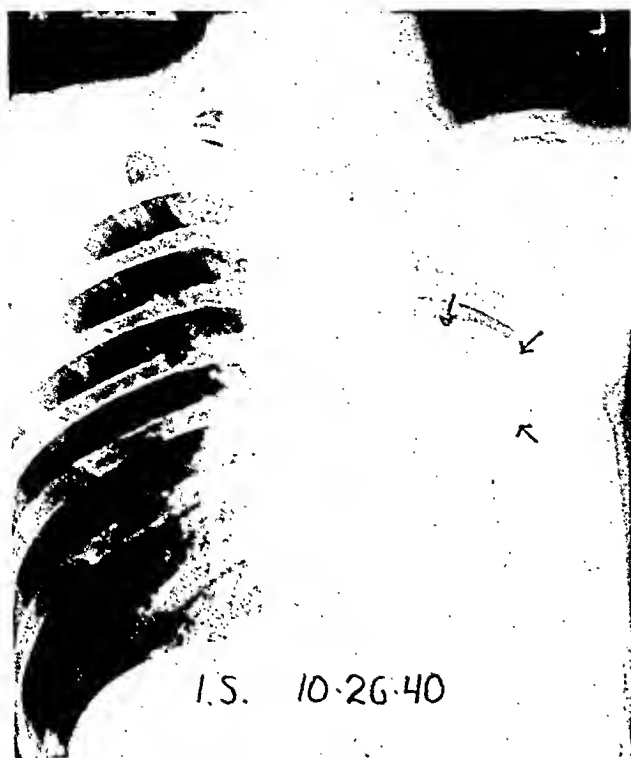
FIG. 6. Case IV reveals large spherical mass measuring 7 cm. surrounding the hilum.

FIG. 7. Case V. This patient was treated by radium introduced with bronchoscope. The residual bronchiectasis and giant cyst are clearly visible.

brown tumor mass with a broad base arising from the left main bronchial wall. The histologic report by one pathologist was "chondroma" and by another, "carcinoma." This slide was also seen by Dr. Shields Warren and his opinion was that it was bronchial adenoma. X-ray (Fig. 6) revealed mottled, increased density throughout the left lung field. There was a large spherical mass, measuring 7 cm., surrounding the left hilum. On November 1, 1940, a left pneumonectomy was performed. Gross specimen revealed that there was at the posterior aspect of the upper lobe a rounded mass at the surface which bulged outward. The hilus showed one bronchus filled with soft, glistening, reddish-brown tissue. Vertical section through the above mentioned mass and converging upon the hilus revealed a circumscribed tumor measuring 6 by 4 by 4 cm. in diameter. Its substance was homogeneous, grayish-tan with foci of hemorrhagic disolorations. It extended almost into the interlobar fissure at the apex of the lower lobe posteriorly. The bronchi contained gray fluid exudate. The pulmonary parenchyma showed the most change in the upper lobe where it was indurated.

Comment. Bronchoscopic biopsy was interpreted differently by three pathologists. This proves that the histologic picture is still disputable. The size of the growth and the patient's age were strongly suggestive of malignancy but the history of four years' duration and the absence of regional metastases to lymph nodes were indicative of a slow-growing, benign lesion.

CASE V. V. R. R., a male aged thirty-four, was admitted to the hospital on January 28, 1941. Seven years prior to admission, following appendectomy, the patient developed a cough productive of greenish sputum. He left the hospital on the twelfth postoperative day, complaining of cough and expectoration. Three months later the patient raised a cupful of bright red blood and on numerous other occasions had blood-streaked sputum. During the subsequent five years the patient was unable to work because of dyspnea. X-ray at that time was interpreted as either tuberculosis or lung abscess. A bronchoscopic biopsy was taken at one of the New York cancer institutes and was diagnosed as malignancy. He was treated by x-ray and radon seed implantation and was bronchoscoped twenty times to bring about adequate drainage. Upon admission the patient showed retraction of the left chest with dullness and bronchial breathing. X-ray showed the left lung to be obscured by a dense, homogeneous shadow, except in the upper portion, where there were numerous, small, saccular shadows of diminished density. (Fig. 7.) The left diaphragm was elevated and the mediastinum was shifted toward the left. The refer-

ring physician, in a letter on February 1, 1941 to Dr. Overholt, stated the following: "As you will note, the reports of the section have been contradictory. Two of our best pathologists have made a diagnosis of bronchogenic carcinoma. Bronchoscopic examination, made by me on two occasions, demonstrated a typical pedunculated tumor about 1½ cm., down below the carina, the surface of which was somewhat lobulated but the mucous membrane over it was apparently intact." Our bronchoscopic examination revealed fibrosis with stenosis of the left main bronchus. There were purulent secretions coming from the lower lobe. In 1937, 1,200 R units were administered from June 10th to July 3rd in thirty applications. On July 17, 1938 one gold radon seed, 1.6 microgram, was inserted into the base of the pedunculated tumor. In March, 1939 more gold radon seeds were implanted but at that time the tumor was still visible and it was removed by electrocoagulation. On February 11, 1941 a left pneumonectomy was performed.

Comment. This case was apparently successful by irradiation, radon seed implantations and electrocoagulation treatment. However, the sequelae brought about by the therapy instituted were as serious as the lesion.

CASE VI. L. W., a female, was admitted to the hospital on January 10, 1942. Eight years ago the patient had moderate hemoptysis. Following this incident, she had frequent colds with hemoptysis. Five years later she suffered from an attack of pneumonia followed by severe pulmonary hemorrhages. She also complained of chest pain, fatigue, anorexia and weight loss. Bronchoscopy revealed a pedunculated, yellowish tumor mass with a smooth surface arising from the middle lobe bronchus and obstructing the main bronchus. The tumor was hypervascular and bled easily. No biopsy was taken because of the fear of severe hemorrhage. X-ray findings were consistent with chronic pneumonitis. A clinical diagnosis of bronchial adenoma with secondary suppurative changes of the right lower and middle lobes was made. A right pneumonectomy was performed on January 9, 1942. The specimen revealed several lymph nodes in fat and fibrous tissue, the largest measuring 1.5 cm. in diameter. On section these nodes were

moderately firm, gray, cellular and contained a large amount of black pigment. The upper lobe was dark purple in color, completely collapsed and soft. The middle and lower lobes were firm and non-crepitant. Within the lung substance, at the ·hilus, a rounded mass, roughly 5 cm. in diameter, was found. This was compressed and bulged into the bronchus in such a manner as to completely obstruct the middle and lower lobe bronchi. The upper lobe bronchus was unobstructed. On sectioning the tumor was seen to be sharply demarcated, encapsulated and slightly lobulated in contour. The bronchi of the lower and middle lobes were dilated. Microscopic diagnosis: "Infiltrating adenoma of bronchus, lymph nodes negative."

Comment. The important points to stress in this case are: the risk of severe pulmonary hemorrhage during bronchoscopic manipulation, the absence of metastatic regional lymph nodes in a patient in the cancer age group with eight years' duration of the disease and a large extrabronchial tumor.

CASE VII. A. J., a male aged nineteen, was admitted to the hospital on October 9, 1942. Duration of the disease was two years. Two years ago the patient had several episodes of hemoptysis, once raising about one-half cupful of bright red blood. However, he remained asymptomatic and well for one year. At that time he developed pneumonia and pleurisy on the right side, associated with wheezing. Roentgenogram (Fig. 8A) showed an area of mottled consolidation in the right upper lobe which extended down to the level of the third interspace. Its lower margin was sharply limited but the area of density was not quite homogeneous. In the shadow there were areas of diminished density which suggested small cavities. The patient was referred to a tuberculosis sanatorium at which time sputum tests were negative for tuberculosis. The patient was treated with pneumothorax. Following the induction of pneumothorax, a complete atelectasis of the right upper lobe occurred. (Fig. 8B.) Because of this finding, bronchial obstruction was suspected and the patient was bronchoscoped. Bronchoscopy revealed a pedunculated mass with gross characteristics of bronchial adenoma arising from the upper lobe bronchus

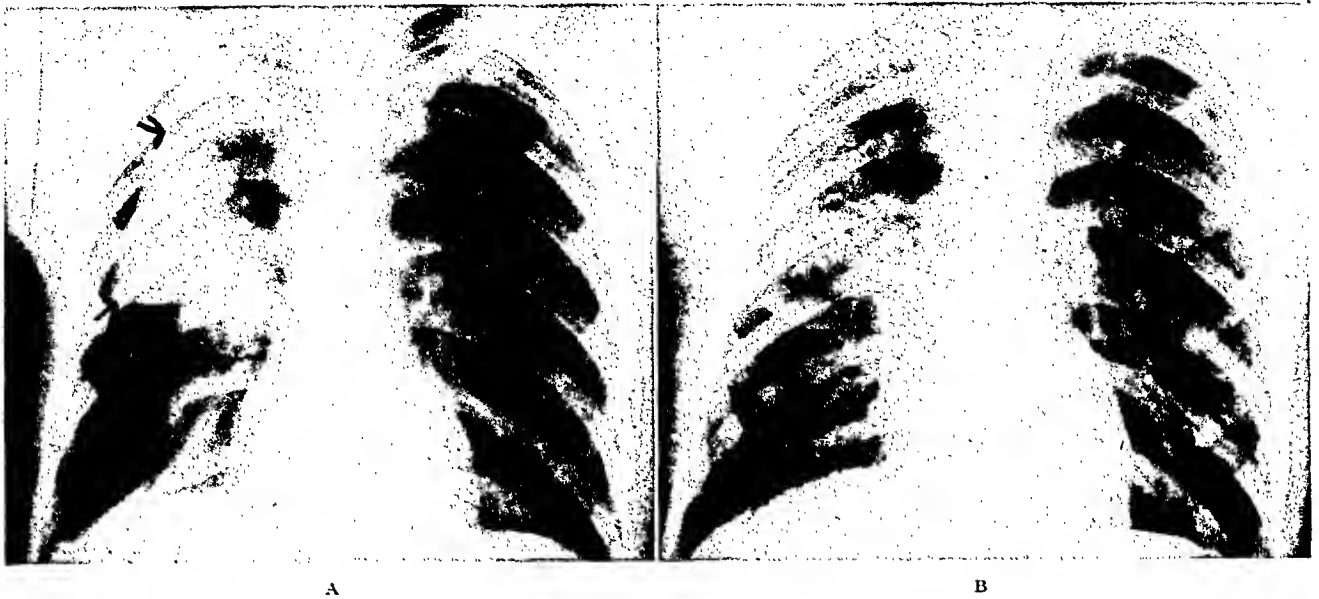A   B

FIG. 8. A, case VII demonstrates an area of mottled consolidation in the right upper lobe which extends down to the level of the third interspace. Its lower margin was sharply limited but the area of density was not quite homogeneous. In the shadow there are areas of diminished density which suggest small cavities. B, same case as A, demonstrates a complete atelectasis of the right upper lobe following the induction of pneumothorax.

and extended centrally toward the carina. The bronchoscopic report was, "bronchial adenoma." On October 11, 1942 a right pneumonectomy was performed. The specimen revealed a small, firm, encapsulated tumor mass in the posterior aspect of the right upper lobe branches, growing into the lung parenchyma and firmly attached to the right main bronchus above the upper lobe orifice. In the right upper lobe branch a pinkish-gray nodule of soft tissue measuring 0.4 by 0.5 cm. was found. Microscopic diagnosis was "submucosa irregularly infiltrated by tumor cells, probably originating from bronchial adenoma."

Comment. In tuberculosis sanatoria patients similar to this one are frequently found because of recurrent hemorrhages. A right pneumonectomy was performed instead of an upper lobectomy because of the infiltrating character of this tumor into the main bronchial wall.

CASE VIII. W. L., a male aged forty-four, was admitted to the hospital on March 29, 1944 because of an acute left empyema which was previously drained. He complained of a chronic productive cough of about one year's duration. The empyema followed an episode of pneumonia on the left side. Following empyema drainage, the patient improved but he continued to have a productive cough, raising about 3 ounces of thick, yellowish, non-odorous

sputum daily. There was no history of wheezing or hemoptysis. Because of the persistent cough, the patient had a bronchogram taken which revealed bronchiectasis in the left lower lobe. No bronchoscopy was performed. A left lower lobectomy was performed. The specimen showed a circumscribed, pale mass 1.5 by 1 by 1 cm. filling the lumen of the basilar branch of the lower lobe. This mass which was smooth appeared to be sharply confined to the lumen
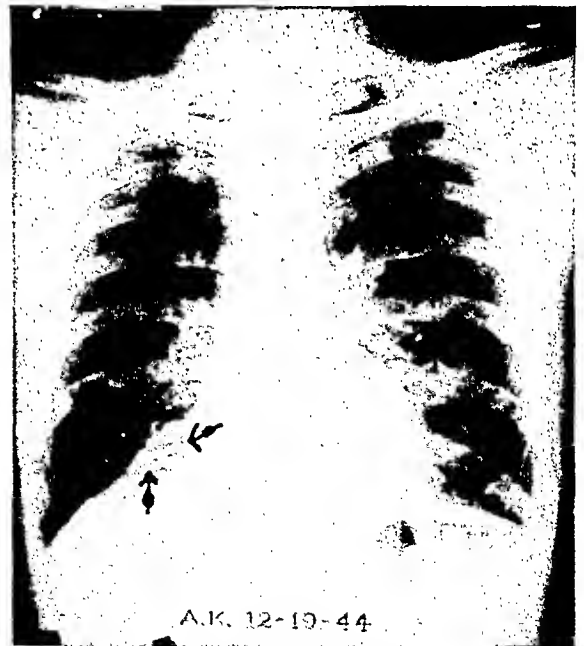

FIG. 9. Case IX demonstrates a triangular area of density at right base in the costophrenic angle. Within this area of density there are small, clear areas which suggest cavities.

FIG. 10. Case IX. A photograph of specimen demonstrating a soft, friable reddish-gray mass bulging from the middle lobe bronchus and causing nearly complete occlusion of the bronchus. This mass extends into the middle lobe parenchyma, measuring 2.4 by 1.2 by 1.6 cm. in size.

of the bronchus. Distal to this obstruction there were dilated bronchi filled with dark, soft material. Microscopic diagnosis was "bronchial adenoma."

Comment. The value of bronchoscopy cannot be overemphasized.

CASE IX. A. K., a male aged fifty-five, was admitted to the hospital on December 20, 1944, with a history of three years' duration, complaining mainly of frequent colds, an average of five episodes each winter with associated cough, temperature and expectoration. Since the previous winter, the patient complained of persistent cough, productive of thick, whitish-yellow sputum occasionally blood streaked for

a few days. He also complained of chest pain, dyspnea and a weight loss of 20 pounds. X-ray (Fig. 9) on admission showed a triangular area of density at the right base in the costophrenic angle. Within this area of density there were small, clear areas which suggested cavities. Radiologic diagnosis was "atelectasis and bronchiectasis of the middle lobe." Bronchoscopy revealed a fungating tumor mass at the level of the right middle lobe orifice which bled easily. The lower lobe bronchus was narrowed to about 50 per cent of its normal size. Bronchoscopic biopsy was reported as "bronchial adenoma, infiltrating in type, potentially malignant." This patient was referred to us with a bronchoscopic biopsy diagnosis of

plasmacytoma. A right pneumonectomy was performed on December 22, 1944.

The specimen (Fig. 10) demonstrated a soft, friable, reddish-gray mass bulging from the middle lobe bronchus and causing nearly complete occlusion of the bronchus. This mass extended into the middle lobe parenchyma and appeared circumscribed, measuring 2.4 by 1.3 by 1.2 cm. in size. The bronchi of the middle and lower lobes showed fusiform dilatation. Microscopic diagnosis was "infiltrating adenoma, metastasizing to two of twenty-one lymph nodes."

Comment. This patient had a pneumonectomy because of regional lymph node metastases, the bronchoscopic picture and age.

CASE X. M. P., a female aged twenty-four, was admitted to the hospital on December 29, 1944. Duration of the disease was two months. The patient was admitted to the hospital complaining of head cold, slight cough, sputum and generalized wheezing. This cough persisted for about one month and became progressively worse. Hemoptysis occurred on three consecutive days, raising large amounts of bright red blood. At that time x-ray was negative. The symptoms subsided for a few days but again appeared. At this time she developed severe chills. X-ray (Fig. 11) then showed atelectasis of the basilar segment of the left lower lobe and emphysema of the superior division of the same lobe. The radiologic impression was atelectasis and emphysema of the lower lobe caused by endobronchial obstruction. Bronchoscopy revealed a tumor mass projecting into the lower lobe bronchus, completely filling the orifice and extending just beyond the orifice of the dorsal division. The tumor was soft and dark red in color. No biopsy was taken because of recent severe hemorrhages. A left lower lobectomy was performed on February 8, 1945. The gross specimen showed atelectasis of the basal division of the lower lobe with emphysema of the remaining segment. A small, smooth, soft, friable mass was found in the lumen of the lower lobe bronchus. Microscopic diagnosis was "infiltrating bronchial adenoma."

Comment. In spite of the short duration of the symptoms the removed lobe revealed permanent, irreparable pulmonary damage
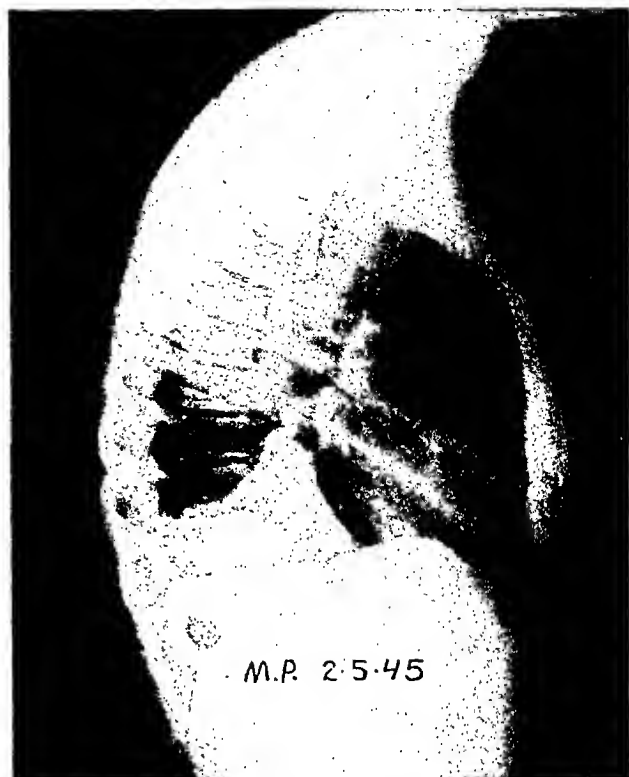

FIG. 11. Case x demonstrates atelectasis of the basilar segment of the left lower lobe and emphysema of the superior division of the same lobe.

in the basilar segment. Bronchoscopic removal was dangerous because of the hypervascularity of the tumor.

CASE XI. P. D., a male aged forty-one, was admitted to the hospital on April 28, 1945. Duration of the disease was eleven years. At onset there was severe pulmonary hemorrhage and pleuritic pain in the right chest. Six weeks prior to admission the patient had a severe hemorrhage which recurred six times in one week. Pneumothorax was instituted to stop the bleeding. Previous to this procedure x-ray Fig. 12A revealed an opacity in the right lower lobe suggesting atelectasis. After pneumothorax the entire right lung became atelectatic. (Fig. 12B.) Upon admission roentgenogram still revealed a residual pneumothorax and there was a single lobe which was well expanded. There was a triangular area of density which represented the collapsed right lower lobe. The middle lobe was not clearly delineated. Bronchoscopy revealed a small, rounded, yellowish tumor mass about the size of an almond in the lower lobe immediately below the orifice of the middle lobe. Many varicose vessels were traversing the surface of this tumor. Biopsy was not taken because of a possible hemorrhage.

FIG. 12. A, case XI reveals an opacity in the right lower lobe suggesting atelectasis; B, same case as A, following induction of pneumothorax because of severe bleeding reveals atelectasis of the entire right lung.

The bronchoscopic impression was bronchial adenoma. On May 4, 1945 a lower and middle lobectomy was performed. During operation a very large bronchial artery was seen and also abnormally enlarged vessels were seen in the pulmonary ligament. Gross specimen revealed a rounded, yellowish, shining structure, 0.5 cm. in diameter, which resembled a lymph node and which was located in the right lower lobe
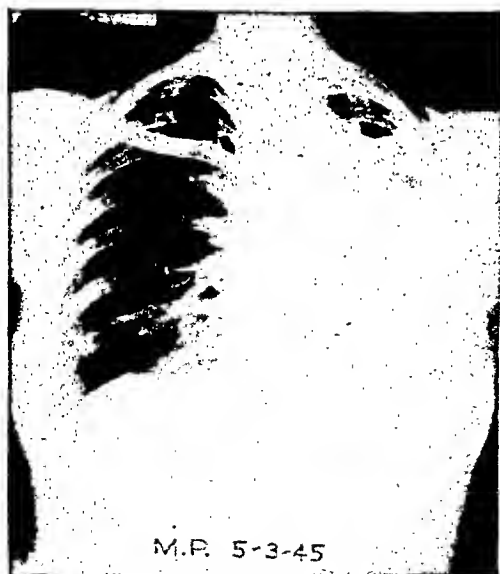

FIG. 13. Case XII reveals opacity of the lower two-thirds of the left lung with multiple clear areas in the left portion of the upper third of the chest.

bronchus. The lower lobe parenchyma was brownish-purple and meaty in consistency. The bronchi showed an irregular dilatation. The bronchus above the growth and lymph nodes was negative.

Comment. Congenital abnormalities were seen in this patient. The middle lobe was sacrificed because of the presence of an entirely fused fissure line.

CASE. XII. M. P., a female aged thirty-six, was admitted to the hospital on March 5, 1945. Duration of the disease was fifteen years. Prior to 1930, the patient developed a severe cold associated with pain in the chest, shortness of breath and slight hemoptysis. At that time she was bronchoscoped and a diagnosis of bronchial adenoma of the left lower lobe was made. The patient continued to have severe hemoptysis. In 1940, piecemeal removal of the adenoma was made by repeated bronchoscopic treatments. In all, she had twelve treatments during 1940 and 1941. Throughout 1942 and 1943 the patient complained of a productive cough, fatigue, a dull ache in the left chest and occasional wheezing. X-ray (Fig. 13) taken upon admission revealed asymmetry of the thorax. The lower two-thirds of the left lung was opaque and there were multiple, clear areas in the left portion of the upper third of the chest. The mediastinum was drawn toward

FIG. 14. Case XIII. Photograph of specimen demonstrating the amount of tumor seen through bronchoscope.

FIG. 15. Case XIII. Photograph of specimen demonstrating the amount of tumor after the bronchus was transected.

the left with elevation of the diaphragm. Bronchoscopy revealed a widened carina and there was traction of the left main stem posteriorly and superiorly with a definite narrowing 1 cm. below the carina. The mucous membrane was of normal appearance above the concentric fibrostenotic area. A left pneumonectomy was performed on May 10, 1945. Gross specimen revealed a small lung with extensive fibrosis and bronchiectatic cavitations throughout, with a smooth, round, firm mass adherent to the anterior surface of the main bronchus. On section this growth was 4 by 3 by 2.8 cm. in diameter and encapsulated and arising from the bronchus. The mucous membrane of the bronchi showed hyperplastic changes with no evidence of a tumor. The microscopic diagnosis was "bronchial adenoma."

Comment. The salient features of this case are bronchostenosis, because of repeated bronchoscopic manipulations, pulmonary parenchymal invasion, early

pulmonary suppuration and absence of regional metastases in spite of the long duration of the disease. This case proved, definitely, the inefficacy of bronchoscopic removal because only a small portion of the tumor which could be seen through the bronchoscope was extirpated. In this respect these tumors are likened to an iceberg which has only a small portion visible above the surface of the water. Pneumonectomy was indicated because of extensive suppurative changes. If this patient were not finally subjected to surgery because of suppuration, this case might have been reported as bronchial adenoma.

CASE XIII.   B. R., a female aged thirty-four, was admitted to the hospital on August 12, 1945. The patient had been coughing for many years. The cough was productive of mucoid material. Four and one half years ago she developed an acute pulmonary process associated

with cough, temperature and blood spitting. Three months later the same clinical picture recurred. X-ray of the chest at that time revealed a triangular, opaque shadow in the right chest above the diaphragm. After a course of sulfa drugs the opacity cleared. In January, 1942 she had a third episode of pneumonitis. In March, 1942 a cholecystectomy was performed but no calculi were found. However, since that time until admission, the patient has had a number of similar attacks associated with hemoptysis. Bronchoscopy revealed the presence of a vascular tumor in the right lower lobe which was diagnosed as bronchial adenoma. Bronchoscopy on August 28th revealed a tumor mass (Fig. 14) in the lumen of the lower lobe just below the spur of the middle lobe. It was palpated and bled very easily. No biopsy was taken because of the marked vascularity. At operation a tumor mass the size of a golf ball was palpated in the posterior medial segment of the right lower lobe. The lobe itself, outside of the tumor mass, appeared normal. One enlarged lymph node was found below the carina and a few smaller ones in the secondary hilum. One of the lymph nodes located close to the bronchi was diagnosed on frozen section as "metastatic node, probably from the infiltrating bronchial adenoma." Because of this a right pneumonectomy was performed. The specimen (Fig. 15) on gross study revealed a rounded, smooth, glistening, pearly, gray mass, $3\frac{1}{2}$ cm. in diameter, protruding from the lower lobe bronchus. It appeared to distend the bronchus causing stretching and thinning of the wall. The tumor margin was discrete and sharp but in a few places it was ill defined and gradually extended deeper into adjacent tissues. Microscopic diagnosis was "infiltrating adenoma with metastases to one of twenty-four lymph nodes. Sarcoidal reaction of the lymph nodes.

Comment. It is noteworthy to mention that pneumonic lesions in the right base may sometimes simulate typical attacks of gallbladder disease. A pneumonectomy was performed in this case because of the presence of one metastatic lymph node.

CASE XIV. E. B., a female aged twenty-five, was well until one year previous to admission at which time she developed three colds in succession, associated with a low grade tem-

perature. X-ray taken at the Middlesex Sanatorium demonstrated atelectasis of the left upper lobe. She entered the hospital complaining of cough, expectoration of mucoid material and fatigue. There was no history of hemoptysis. On January 17, 1946, bronchoscopy showed the upper lobe orifice to be displaced upward with visualization of the lower spur. No abnormalities were seen. A diagnosis of bronchial obstruction, left upper lobe, was made, most likely due to tumor. Bronchographic studies showed an amputation of the upper lobe bronchus. On January 22nd a total pneumonectomy was performed. At operation a completely atelectatic upper lobe was seen and approximately $\frac{1}{4}$ cm. above the origin of the upper lobe orifice a round, solid, yellowish smooth mass, about the size of a walnut, was palpated. The upper lobe was airless, indurated and approximately two-thirds of its normal size. Enlarged lymph nodes were found in the mediastinum and fissure line. The lower lobe was emphysematous. It was technically impossible to do an upper lobectomy because of the fused fissure line and the presence of many enlarged lymph nodes in the secondary hilum. Microscopic report was "bronchial adenoma, lymph nodes negative for metastasis."

Comment. Adenoma in this case originated in the lobar bronchus at the level of its divisional branches. Because of the location of this tumor, a pneumonectomy was performed. A lobectomy was technically impossible because of the completely fused fissure line.

CASE XV. F. R., a male aged twenty-seven. In 1938 the patient developed pneumonia on the right side. Since then, he has had recurrent episodes of pneumonia on the same side followed by chronic cough and expectoration. X-rays taken on three different occasions during the past year demonstrated atelectasis of the right lower lobe. X-ray showed an area of opacity in the right base suggesting atelectasis. Bronchoscopy on March 27, 1946, showed approximately 1 cm. below the middle lobe orifice a tumor mass projecting from the lumen of the basilar bronchus. The mass was yellowish in color, had a smooth surface and bled easily upon being touched. Bronchoscopically, the mass had all the characteristics of a bronchial adenoma and a biopsy was not taken because of

the danger of hemorrhage. A right middle and lower lobectomy was performed on April 1, 1946. The right lobe contained a tumor mass the size of a tangerine which was felt attached to the basilar bronchus. Biopsy of the lymph nodes which were present close to the lesion were negative.

Comment. This case illustrated again that the clinical manifestation of bronchial adenoma may be those of chronic pneumonitis, demonstrating the importance of early bronchoscopic examination. A middle and lower lobectomy was performed because the tumor mass was found to be too close to the middle lobe orifice.

CONCLUSION

We have presented fifteen cases of bronchial adenoma cured by pulmonary resection, ten by pneumonectomy and five by lobectomy. Bronchial adenoma is a disease that should be treated either by lobectomy or by pneumonectomy.

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ANALYSIS OF ACUTE CRANIOCEREBRAL INJURIES*

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APPROXIMATELY 6 per cent of all the battle wounds that occurred in the Mediterranean theater in 1944[1] involved the head (exclusive of maxillo-facial injuries), whereas, in a series of 1,000 battlefield autopsy examinations it was found that 40 per cent had incurred head wounds of sufficient severity to cause death.[2] This disproportionate immediate mortality from head wounds indicated that early reparative surgery is imperative in this type of injury. The first surgeon to give other than supportive treatment to these patients was the surgeon in a field or evacuation hospital. Throughout the campaign an attempt was made to maintain a neurosurgeon with each forward hospital. This is a report of the author's experiences while acting as the neurological surgeon in a 400 bed evacuation hospital where 4.9 per cent of the hospital admissions had craniocerebral injuries.

GENERAL EVALUATION

At the time of admission the patient with a craniocerebral wound was evaluated much as any other patient. The cardiovascular status was of prime importance. The most useful adjunct for stabilizing the vascular system was found to be whole blood. During the war years of 1942 and 1943, plasma was the substitute of choice, but, with experience, whole blood was found to be of more value. An average of 900 cc. of whole blood was given to each patient with a craniocerebral wound.

The patient was then evaluated from a neurological standpoint. It was found that certain signs indicated a poor prognosis. Pupils that were dilated and fixed to light were significant; five out of eight patients with this sign expired. Patients who main-tained extensor muscle spasm in two or more extremities (decerebrate rigidity) offered a poor prognosis; five of the six patients with this sign expired. If consciousness had been maintained at all times, the outlook for survival was certain; there were no deaths in 270 patients. If the patient had been unconscious and had regained consciousness, the prognosis was also good; there were no deaths in eighty-two patients. But if the patient was immediately unconscious following the injury and remained so until the time of operation, the prognosis was poor; twelve out of twenty-nine patients expired. Hemiplegia or abnormalities in deep tendon reflexes (other than spastic paraplegias) were not found to be of significance in the prognosis of mortality; however, reflex changes were valuable in estimating the amount and site of cerebral damage. Patients with diffuse cerebral damage resulting from direct contact with missile or bone fragments or from concussion offered a poor prognosis. The amount of cerebral tissue that herniated through the cranial wound bore very little relationship to the prognosis. Small wounds often permitted no herniation of cerebral tissue nor leakage of blood, consequently, patients with this type of wound often suffered from unduly increased intracranial pressure.

Patients were then evaluated with regard to their general physical status. Associated injuries of the chest, abdomen or extremities were common. Wounds of the chest and/or abdomen took preference over the craniocerebral wound unless it was evident that the patient was suffering either from markedly increased intracranial pressure or a large intracranial vessel was bleeding. The size of the cranial defect was impor-

tant since a large or grossly contaminated wound required longer operative procedures.

TREATMENT

The patients were observed and given supportive therapy until the vital signs became stabilized, usually sometime within twelve hours, and operative repair was performed at this optimum time; but if a continually downhill course ensued, a craniectomy was immediately performed. It was found that the period of stabilization could be prolonged as long as seventy-two hours without increased danger of infection, but after this period the chance for postoperative infection increased rapidly. Obviously moribund patients were given the benefit of surgery even though no improvement resulted from supportive therapy.

Preoperatively postero-anterior and lateral roentgenogram examination of the skull was always made but not stereoscopic examination because it was found to give very little additional data.

Scalp Wounds. There were 230 patients treated for scalp wounds. These varied from small lacerations to almost complete avulsion of the scalp. Patients with scalp wounds only seldom gave a history of unconsciousness (eleven of 230 cases). During periods of heavy casualties, these wounds were treated by the general surgeons and the neurosurgeon was called only if complications were found. The patients were taken to the operating room, the area around the wound was shaved, cleansed with soap and water and painted with mercresin solution. Strict toilet was considered to be imperative. Procaine solution (1 per cent) with adrenaline was infiltrated around the laceration, or if over the forehead, a supra-orbital nerve block rendered the wound anesthetic. The wound was excised *en toto* and the defect washed with saline solution. The galea and skin were always closed in layers with silk sutures. Skin sutures were removed on the third postoperative day in wounds over the forehead but on the fifth postoperative day in wounds above the hairline.

Simple Depressed Fractures. There were fifteen patients who presented simple depressed skull fractures (closed injuries). Patients admitted to the hospital because of closed head injuries, with or without simple linear fractures, are not included in this series (156 patients). A careful evaluation was made of the size of the depressed fracture and of the neurological signs and symptoms. If the depression were small and the patient asymptomatic, no attempt was made to elevate the fragments. There were eleven patients in this category. These patients were closely observed for later occurrence of neurologic signs. In none of them was subsequent surgery indicated.

In the other four patients craniectomies were performed, because of the large size of the depressed bone or persistent neurological signs such as a hemiplegia. In two of the four, there were lacerations of the dura with a small amount of pulped cortical tissue underlying the fracture. In the other two patients large extradural hematomas were found, evacuated and the bone fragments elevated. The technic used was to place a burr hole adjacent to the depressed fragments and to elevate the fragments with a periosteal elevator. In two of the patients, it was necessary to remove some of the depressed fragments and the defect created by the removal of these fragments was used instead of a trephine hole to introduce the periosteal elevator. A tense non-pulsating dura was considered to be an indication of subdural injury. Dural lacerations were closed with silk sutures.

Compound Depressed Fracture (Non-missile). There were ten patients with compound depressed fractures that were not due to high velocity missiles. The treatment of these patients was similar to that of simple depressed fractures that required operation. These wounds were also carefully débrided, the dura closed and the scalp sutured in layers. In eight of these patients, there were dural lacerations and damage to the underlying cortex but in none of them was there evidence of hematoma.

Compound Depressed Fractures (Missile). There were 126 patients with compound depressed fractures due to high velocity missiles. There was penetration of the dura in 100 of them. The treatment given to the twenty-six patients without dural

TABLE I
MISSILE WOUNDS WITH DURAL PENETRATION

Type of Injury	No.	Mortality	
		No.	Per Cent
Penetrating.................	86	7	8
Perforating.................	14	5	36
Side-to-side (4)............	...	(2)	(50)
End-to-end (10)...........	...	(3)	(30)
Total.................	100	12	12

penetration consisted of excision of the wound of entry, removal of all contaminated bone and closure of the wound without drainage. The 100 patients with missile wounds in which the dura was penetrated were the greatest problem. Table I shows that the majority were due to penetrating rather than perforating missiles. The higher mortality rate for the perforating wounds was probably due to the greater extent of damage concomitant with the higher velocity of the missile. Table II shows the site of entrance of the missiles and the mortality rate. The frontal lobe was most often the site of injury; in fourteen patients both cerebral hemispheres were involved by the missile tract. The higher mortality in these patients with wounds of the frontal lobe may be due to the fact that in all six the missile had penetrated either one or both ventricles or had lodged deeply in the hypothalamic area.

There were ten transventricular wounds; six of these patients expired. In all six, there was concomittant severe damage to the paraventricular structures. None of these patients with transventricular wounds died as a result of intraventricular hemorrhage, perhaps because after operation there was always an exit for the blood other than

through the foramen of Monroe. None of them developed a suppurative ventriculitis.

The frontal sinus was involved in twelve patients of whom four died, and the mastoid was involved in four patients, one of whom died because of a concomit-

TABLE II
MISSILE WOUNDS—SITE OF ENTRANCE

Site of Entry	No.	Mortality	
		No.	Per Cent
Frontal...............	20	6	(30)
Temporal................	18	1	(5.5)
Parietal................	55	4	(7.2)
Occipital..:..............	3	1	(33)
Cerebellar..............	4	0	0
Total.................	100	12	(12)

tant dural sinus injury. The incidence of infection was not increased due to sinus involvement. There were two patients with rhinorrhea following craniectomies for injuries of the frontal sinus, both of whom ceased draining spontaneously within seven days postoperatively. At the time of débridement all loose tissue in the sinus was removed, but intact sinus membrane was not disturbed because it was thought that this membrane acted as a barrier against infection.

In seven patients, a major dural sinus was involved, the sagittal in four, the transverse in two and the sigmoid in one patient. Only one of these died and this was a patient with injury to the sigmoid sinus. Unfortunately in this patient there was an anomalous partial atresia of the left transverse sinus while it was the right sigmoid that had been lacerated. A postoperative thrombosis of this unpaired right sigmoid sinus resulted in death.

At operation, six subdural and two extradural hematomas were found. All of these were on the side of the wound of entrance.

Preoperatively a neurological examination was always done. In Table III, the patients are grouped according to the most

prominent neurological sign observed at the preoperative examination, i.e., a patient with a paralysis of the arm and reflex changes in the leg is grouped under paralysis—arm. Patients with involvement of the reflexes of the leg, arm and

TABLE III
MISSILE WOUNDS—NEUROLOGICAL CHANGES

Neurological Change	Arm	Leg	Arm Leg	Arm Leg Head	Total
Reflex	2	10	32	24	68
Paresis	2	..	12	..	14
Paralysis	4	..	4	..	8
Total	8	10	48	24	90

cranial nerves are grouped under reflex—arm, leg and head. In ten patients no abnormalities were found in the neurological examination. Five patients had an aphasia preoperatively; the aphasia persisted after operation. Two of the patients had an anomia and three had a mixed type of aphasia. In six patients, there was involvement of the retrobulbar visual system.

Table IV reveals the number of patients who were unconscious. A patient was considered to be unconscious only if he did not respond in any visible way to a moderately noxious stimulus (pin prick). Sim-

TABLE IV
MISSILE WOUNDS—STATE OF CONSCIOUSNESS

Site of Injury	Unconscious	Conscious	No. of Patients
Frontal	17	3	20
Temporal	15	3	18
Parietal	45	10	55
Occipital	3	0	3
Cerebellar	4	0	4
Total	84	16	100

ply being unaware of his environment was not considered unconsciousness. There seems to be no specific area of the brain that, when injured, predilected to a state of unconsciousness.

There were twelve deaths (12 per cent) in this group of patients with dural penetration by a missile. Clinical evidence of increased intracranial pressure was present in eight of them, pulmonary edema and/or pneumonia in six, whereas evidence of hypothalamic damage was present in

TABLE V
DATA ON PATIENTS WHO EXPIRED FOLLOWING MISSILE WOUNDS WITH DURAL PENETRATION

Hours from Injury until Hospital Admission	Hours from Injury until Operation	Days from Injury until Death
6	24	9
2	6	20
2	24	8
3	18	14
12	18	2
13	16	3
9	18	6
1	5	2
17	29	10
18	27	28
22	31	23
13	18	5
Average 9.1 Hours	19.5 Hours	8.0 Days

two. In none of the eight patients with evidence of increased intracranial pressure was there evidence of a space consuming lesion at autopsy. Multiple scattered petechial hemorrhages throughout the gray and white matter was a frequent finding. One patient died as a result of a postoperative wound infection. He expired from meningitis on the twentieth day.

Table V shows in these patients who expired, the period of time between injury and hospital admission, operation and death. All patients who expired were rendered immediately unconscious by the missile and did not regain consciousness up to the time of operation.

Preoperatively patients with dural penetration were treated to obtain maximum improvement. The usual methods of combatting shock were carried out. Oxygen was administered by mask or under pressure via an intratracheal tube. The frequently observed pulmonary edema was treated by positive oxygen pressure and

also by repeated tracheal aspirations. Codeine rather than morphine was used to relieve pain. Morphine was not used because of its depressant action and its tendency to obscure neurological signs. The anesthetic of choice was a solution of 1 per cent novocaine containing adrenalin. To unusually restless patients sodium pentothal or avertin fluid was given per rectum or sodium pentathol solution intravenously. These drugs were given to quiet the patient and were not given in analgesic dosages.

The wound of entry was excised and the bone defect enlarged to approximately 4 by 5 cm. except occasionally when larger defects up to 8 by 18 cm. in size were found necessary to visualize the entire dural and brain wound. The contaminated dura was excised, macerated brain tissue was excised until healthy tissue was encountered, and all indriven bone fragments were removed regardless of their location. Metallic fragments that were small and deeply located were not removed unless an evident tract led down to them, but large metallic fragments were always removed even if it were necessary to perform a second craniectomy over the site where the missile had lodged. Tracts were followed across the midline if necessary in order to obtain an adequate débridement. "Lobectomy necessitans" were required oftentimes because of the extent of the damaged brain. An attempt was always made to close the dura; in 46 per cent of the patients it was necessary in order to obtain closure to use some form of graft and for this fascial grafts obtained from the galea, temporal fascia or fascia lata were used. The galea and skin were always closed in separate layers without drainage. Oftentimes a Z-plasty or rotation flaps were required to close the scalp defect.

POSTOPERATIVE CARE

Special postoperative care was found to be important. The total fluid intake was maintained at less than 3,000 cc. daily. Whole blood was the substitute of choice for intravenous therapy. To promote cerebral venous drainage, the head was elevated except for the cerebellar wounds in which the "coma position" as described by Dandy was used. A careful record of pulse, blood pressure and respiratory rates was made and used as a guide in the postoperative care. The bronchial tree was maintained as free of fluid as possible by repeated aspirations and, if necessary to insure a non-obstructed airway, an intratracheal tube was left in place for thirty-six to forty-eight hours. Intramuscular penicillin 20,000 units, every three hours, was given for seven days postoperatively, and whenever a wound extended into a ventricle, 20,000 units of penicillin was instilled into the wound. No sulfa therapy was instituted at the time of operation or later unless penicillin failed to control an infection.

SUMMARY

A report on 381 craniocerebral injuries seen in a forward evacuation hospital is presented. The majority (230) were scalp wounds. The greatest therapeutic problem was presented by the 100 patients with compound depressed fractures with penetration of the dura by a missile. Abnormalities found by the neurological examination are discussed.

Early reparative surgery was performed. A period for stabilization of the vascular system preoperatively was found to be valuable. At the time of craniectomy, the brain tract was thoroughly débrided of all indriven bone fragments. Tight closure of the dura was performed.

There was a mortality rate of 12 per cent in those cases with dural penetration. This included all deaths occurring prior to evacuation of the patient to the United States.

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OSGOOD-SCHLATTER'S DISEASE

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OSGOOD-SCHLATTER'S disease of the tibial tubercle, judging from the usual textbook description and from the relatively few reports in the English literature in the last twenty years, is a fairly rare entity, having a generally typical clinical manifestation, a varied roentgenographic appearance and a frequent defiance for sundry therapeutic measures. This paper was inspired, therefore, because of the discovery of thirteen cases in the short period of nine weeks at a large Naval recruit training center, and because it has become apparent that the roentgenographic appearance of the lesion allows for classification in a manner hitherto unsuggested by investigators of this problem.

ETIOLOGY

It is the common observation among all men dealing with cases of Osgood-Schlatter's disease that trauma to the tibial tubercle is the most frequent etiologic factor found in the patient's history. This trauma may be direct as a result of a fall on the knee, or it may be indirect from violent quadriceps contraction as in running or in kicking. There are, however, a certain number of cases in which a history of trauma is not obtainable. It also seems difficult to understand how the extensive x-ray findings of Schlatter's disease could develop in the few days elapsing between the date of the trauma and the date of x-ray in those few patients who report to their physician immediately after trauma and the onset of symptoms. For these and other reasons the etiology remains a matter of dispute. O'Kane[3] believes there may be two distinct groups of cases, one purely traumatic, the other a case of new bone

formation in the patellar ligament. Meyer[4] summarizes most theories as follows: (1) developmental etiology; (2) endocrine dyscrasia; (3) infectious theory (Pease,[17] in 1934, reported a case in which Streptococcus viridans was cultured from a specimen of the tubercle removed at operation); (4) injury. Cole[7] points out that the disease usually appears at puberty and believes it to be a result of stress applied from the quadriceps to the tubercle of the tibia via the patella and infrapatellar ligament at a time when the tubercle can least stand the strain. This may be associated with trauma but, as Cole points out, 66 per cent of his twenty-four patients had bilateral lesions and in their histories trauma had been incurred only on one extremity.

We do not attempt the addition of any new theory to the etiologic riddle but merely state, as have many before us, that the majority of patients date the onset of complaints from some traumatic episode.

PATHOLOGY

Cole[7] and Uhry[10] have reported microscopic studies of sections removed from the area of the tibial tubercle at operation in cases of Osgood-Schlatter's disease. These reports show that early in the disease process there is hemorrhage between the two separated elements in the tubercle. This clot is then gradually organized by the invasion of fibroblasts and connective tissue fibers with the formation of scar tissue. In other sections the two bone plates were seen to be separated by fibrocartilage at which time the sections appeared as a typical fracture with non-union. Cole[7] further describes on gross examination an increased vascularity of the tendon at its attachment to the tubercle, and micro-

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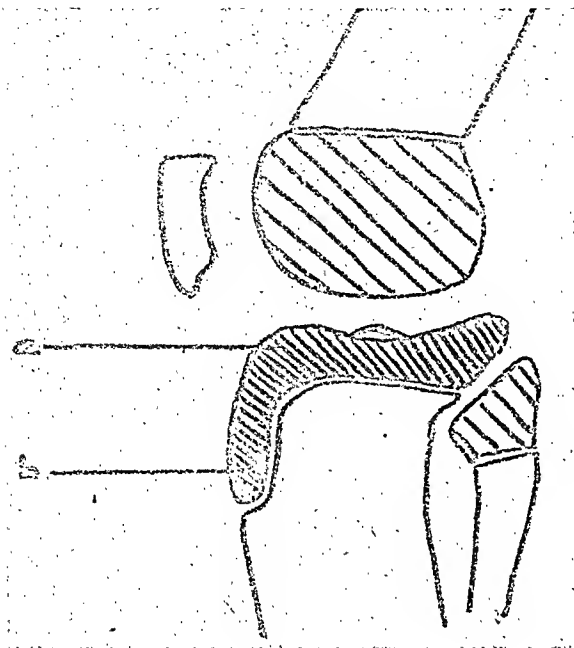† Formerly Lieut., M.C., U.S.N.

FIG. 1.

scopically a roughening and irregularity of the line of attachment of the tendon and tubercle. In contrast, the line of union of tubercle and tendon is smooth and even in the normal individual.

FIG. 2. A, epiphyseolysis.

SIGNS AND SYMPTOMS

The diagnosis is often possible from the history and physical findings alone and can be easily confirmed by x-ray examination. As previously stated, the most frequently encountered report from the patient is that following some trauma to the knee (or knees) he noted the onset of pain in the region of the involved tibial tubercle. This pain may be present continually in the far advanced cases, but early in the course of the disease it is present only when a pulling force is transmitted to the tubercle from the quadriceps via the infrapatellar ligament, as in standing in the military position of "attention," kicking a football, running or doing calisthenics. In addition, the patients often complain of pain over the tubercle on kneeling or direct pressure. Objective findings are limited to the area of the tubercle where there is always a moderate to marked degree of soft tissue swelling and bony irregularity. In no case
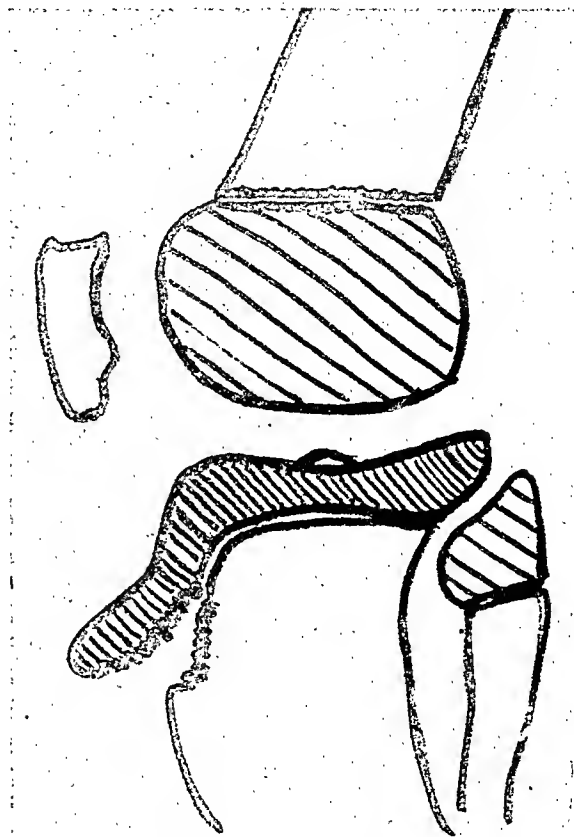
was this enlargement not immediately discernible. Some authors have observed signs of inflammation over the tubercle with redness, increased local heat, tenderness and swelling. (In our series no patient showed any evidence of such an acute process.) There was pain consistently on pressure over the tubercle. If one asks the patient to extend the knee and maintain the quadriceps in contraction, attempting to move the patella laterally will cause pain in the region of the tubercle. In the long standing case it may even be impossible for the patient to contract the quadriceps without pain in the area of the tubercle.

X-RAY FINDINGS

The roentgen anatomy of the proximal portion of the tibia is relatively simple. The proximal tibial epiphysis, as seen in the lateral aspect (Fig. 1a), has a tongue-shaped inferior projection (Fig. 1b) on the anterior surface, representing a portion of

FIG. 2. B, epiphyseolysis (retouched).

the tubercle. The greater portion of the tibial tubercle is included by the epiphyseal line. Ossification of the proximal tibial epiphysis appears just before or just after birth; downward ossification occurs (Osgood) or a secondary center of ossification appears in the tubercle (Schlatter) and the epiphyseal portion of the tubercle begins to ossify at approximately eleven years. Fusion of the epiphysis to the shaft occurs usually by nineteen years. When ossified, the tubercle is a low, irregular elevation on the anterior aspect of the bone 3 to 5 cm. below the articular surface. The superior portion is fairly smooth, the site of attachment of the ligamentum patellae; the inferior portion is roughened, more subcutaneous and there may be a ridge or a depression between it and the superior portion. A groove often delimits the upper edge of the superior portion.

The term "epiphysitis" often applied to the condition described by Osgood in 1903 and Schlatter in 1908 is an inappropriate one since it indicates an inflammatory process. A similar disadvantage holds for "osteochondritis" while "osteochondrosis" is better. For this reason no term thus far used is completely satisfactory.

True Osgood-Schlatter's disease, if the term is to remain in medical language, should be reserved for a necrosing process of the tibial tubercle, with the deposition of osteoid tissue following necrosis and re-ossification after healing.

The roentgenographic manifestations of Osgood-Schlatter's disease have been variously described. Bosworth[6] describes two acute stages and a chronic stage; the former may be evident by haziness about the epiphysis with extraneous calcification superficial to the tubercle, periosteal elevation or partial epiphyseal separation, and the latter by separate bony structures in the patellar ligament or the tubercle area. Cole[7] finds enlargement of the patellar
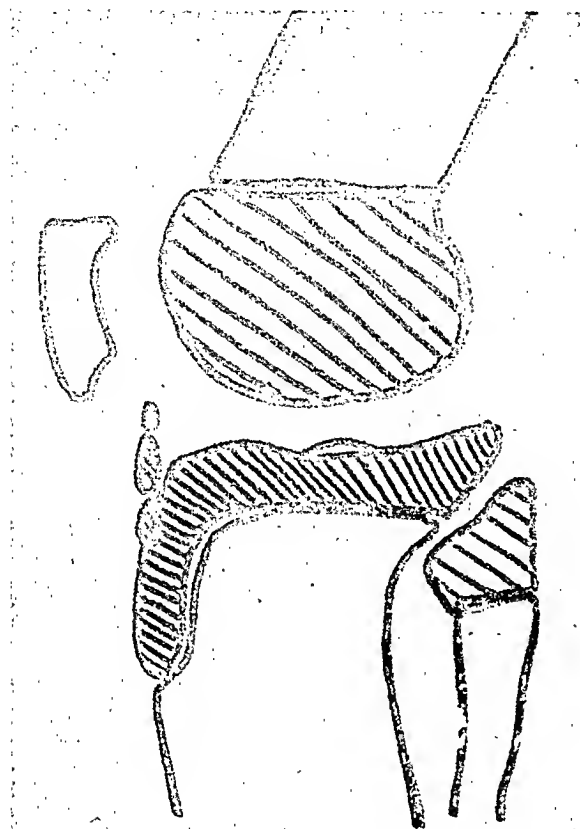
FIG. 3. A, fragmentation.

ligament insertion the first x-ray change. Sutro and Pomeranz[15] describe ligament thickening and bony islands in the ligament.

It has become apparent, therefore, that roentgenographically what is known as Osgood-Schlatter's disease may be one of three distinct processes, all of which probably have the same etiologic factors operating. The first of these, simple epiphyseolysis without fragmentation of the epiphysis, is shown in Figures 2A and B. Fragmentation and necrosis may follow, but the primary process is an epiphyseal separation. This occurred twice in our series. Type 2 is a fragmentation of the epiphysis, with or without epiphyseolysis, but without roentgenographically demonstrable aseptic necrosis of the avulsed fragments. The primary process here is avulsion-fracture of the tubercle. (Fig. 3A and B.) This occurred ten times in our series, two with epiphyseolysis and eight without. The third type, that least frequently seen roentgenographically, corre-sponds to the usual description of Osgood-Schlatter's disease. (Fig. 4A, B and C.) There is fragmentation of the epiphysis with or without epiphyseal separation and with fragment necrosis. This type occurred only twice in our series. In these latter two types the patella is frequently elevated due to the partial rupture of the insertion site of the ligamentum patellae.

It is at once obvious that epiphyseolysis or fragmentation may be acute processes, the result of recent injury (or whatever the initiating factor may be) or an old process with or without clinical symptoms. The x-ray criteria of activity would then depend on the appearance of the adjacent bony margins at the separated epiphysis, or in serial roentgenograms, changes in bone density, developing lacunae or trabeculae alteration in the fragments or changing size of the fragments. Overgrowth of adjacent bone may occur as a secondary manifestation; thickening of the adjacent soft tissues is frequently present. The "extraneous calcifications" in the tubercle area are, for the most part, actually avulsed particles from the epiphysis.

Thus, any of these three types may be acute (active) or chronic (active or inactive) depending both upon the clinical as well as the roentgenographic findings. With these facts in mind, the following classification of what is generally termed Osgood-Schlatter's disease is suggested:

Type 1. Tibial tubercle epiphyseolysis
Type 2. Tibial tubercle fragmentation
        (a) With epiphyseolysis
        (b) Without epiphyseolysis
Type 3. Tibial tubercle necrosis
        (a) With epiphyseolysis
        (b) Without epiphyseolysis

The differential diagnosis by x-ray offers little problem. When tubercle necrosis occurs, a primary pyogenic infection may be suspected. This is a rare occurrence since that disease is usually primary in the metaphysis with secondary involvement of the epiphysis and adjacent joint. The clinical findings and serial films serve to differentiate. Congenital syphilis often in-

FIG. 3. B, fragmentation.

volves multiple bones and serologic studies aid in differentiating. Acquired syphilitis osteomyelitis and tuberculous osteomye-litis are medullary infections, involving cortical, periosteal and epiphyseal struc-tures secondarily; clinical, laboratory and serial roentgenographic studies would lead to the correct conclusion. A bone tumor would rarely confuse. In the absence of tubercle necrosis, when only simple epi-physeolysis or tubercle fragmentation are present, no difficulties in diagnosis are encountered.

CASES

It will be seen from the accompanying tables that all but two of our patients were seventeen years of age. (Table 1). The cases are classified as to the type of onset, duration of symptoms, the knee involved, presenting symptoms, x-ray findings and those seeking medical attention prior to their naval service. Nine of our patients (69.2 per cent) gave a history of trauma. In only one of these was that trauma de-scribed as indirect. This patient dated the onset of symptoms to three months prior to naval service when he had picked up a heavy bundle and had noted pain in both knees. Examination demonstrated bilateral disease.

The average duration of symptoms was 3.5 years and of these thirteen coming under observation in only four (30.8 per cent) had these symptoms been severe enough for them to seek medical advice. One may, of course, wonder if many men would not report to a free navy sick call with symptoms the severity of which would never take them to a doctor were they in civilian life. However, it was our opinion that in the majority of cases these men had legitimate symptoms and according to their statement these symptoms were consider-

ably increased by the working, drilling and exercising on the cement drill fields of the training center.

In Table 1 the symptoms recorded are those which the patients experienced in service. It will be seen that, as expected, left-sided localization of the process. In light of the relatively few recorded cases, however, we believe that if any true evaluation of the frequency of unilateral involvement is to be made one must note any preference of the disease for one side.

TABLE 1

Age	Type of Onset	Symptoms	Years Duration	X-ray	Sought Rx Before	Knee Involved
17	Fall	Pain on marching	3–4	Fragmentation Epiphyseolysis	No	Right
17	Insidious	Pain on marching	3–4	Necrosis Epiphyseolysis	No	Left
19	Football	Pain on marching	4	Fragmentation Epiphyseolysis	No	Left
17	Football	Fatigue marching and calisthenics	3	Fragmentation Epiphyseolysis	Operation and cast	Bilateral
17	Insidious	Painful marching and calisthenics	4	Fragmentation	No	Right
17	Fall, bicycle	Pain, calisthenics	1½	Fragmentation Epiphyseolysis	No	Left
17	Insidious	Painful marching and calisthenics	3	Fragmentation Epiphyseolysis	No	Left
17	Football	Pain stading at attention	3	Fragmentation Epiphyseolysis	No	Right
17	Fall, bicycle	Pain on marching	3	Epiphyseolysis	No	Bilateral
18	Insidious	Weakness marching and calisthenics	3	Fragmentation Epiphyseolysis	No	Left
17	Carrying bundle	Pain on marching	3 mo.	Epiphyseolysis	Yes, no Rx	Bilateral
17	Fall	Pain standing at attention	4	Fragmentation Epiphyseolysis	Yes, no Rx	Right
17	Fall	Painful marching and calisthenics	9	Necrosis	Yes, no Rx	Left

those activities which demand contraction of the quadriceps cause the most discomfort.

Although the literature records no predilection of the disease for one knee, in our cases the left knee alone was involved in 46.1 per cent of the cases, the right alone in 30.8 per cent and both knees in 23.1 per cent. We believe this is too small a series to allow the conclusion that there is any

TREATMENT

Most authors are of the opinion that conservative treatment with posterior splints, cylindrical casting, taping or ace bandaging over the tubercle, and/or bed rest is the treatment of choice in the early case. The consensus favors a trial of such treatment before more radical measures are at-

tempted. There is also a more or less general agreement that in the cases in which symptoms persist beyond four to six months under conservative therapy, operative intervention should be employed if early "cure" is to be accomplished.

TABLE II

Cases No.	Improved		Not Improved		Made Worse		Unable to Follow	
	No.	%	No.	%	No.	%	No.	%
13	6	46.1	2	15.4	2	15.4	3	23.1

In our series conservative therapy was employed throughout. An attempt was made to follow these men at monthly intervals with a discussion of the subjective improvement and follow-up x-rays. The follow-up period was limited to the short span of six to eight weeks in which the trainee was on the station and in no instance did this actually exceed six weeks. In no case was there any x-ray evidence of regression of the lesion on the follow-up film nor was any expected. Nevertheless in Table II one will note that subjective improvement was obtained in 46.1 per cent of the patients under treatment, 15.4 per cent showed no improvement, 15.4 per cent were made worse and in 23.1 per cent we were unable to make any follow-up studies.

Figure 5 is an illustration of the type of therapy employed in all our cases. It must be remembered that it was impossible to employ bed rest or in any way restrict the activity of the men in this series. Our patients passed through their "boot" training with no lightening of their duties in any respect. In such a case one will realize that it is impossible to restrict completely the motion of the involved joint and expect a man to march with his company, do calisthenics and the other physical activity necessary to keep up with the rest of his unit. Our therapy was, as in all conservative therapy, directed toward relieving the pull of the quadriceps on the tibial
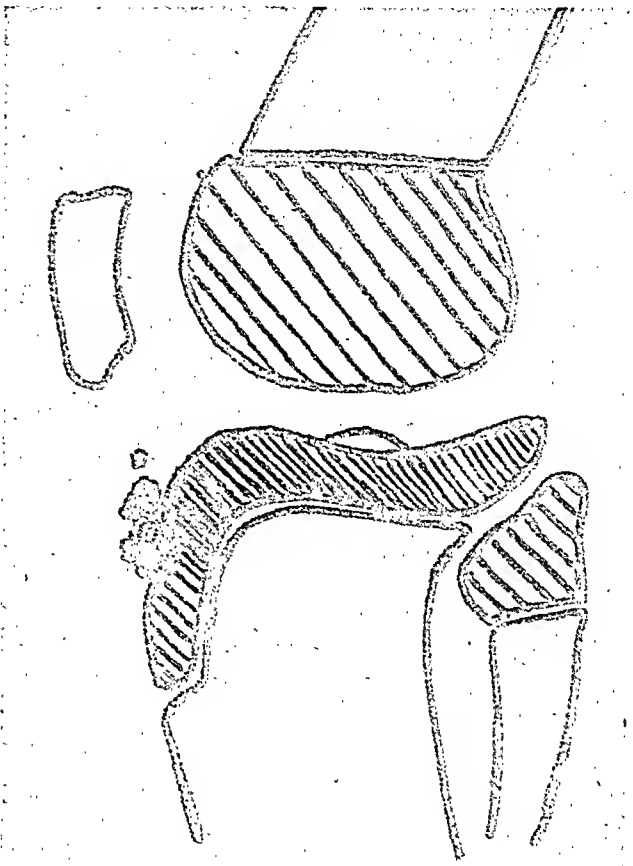

FIG. 4. A, necrosis.

tubercle. This was attempted with cross strapping as diagrammed, and in some cases the employment of a circular ace bandage around the tubercle area. The greatest difficulty was the fact that in the vigorous exercise necessary in the man's daily activity the taping would seldom stay in place for more than forty-eight hours. In all cases, however, the tape was continually reapplied as long as the skin irritation did not form a contraindication. In light of the aforementioned difficulties encountered in treatment we believe that an observed 46.1 per cent improvement in this group is of some significance.

The tape was applied in the following manner: With the patient sitting, the leg was placed in relaxed extension by allowing the heel to rest on a low stool without supporting the knee, after shaving the leg and applying compound tincture of benzoin to the skin. The cross tapes were applied from above downward with the traction force being applied downward in an attempt to relax the tension on the infrapatellar tendon

FIG. 4. B, necrosis; C, necrosis (lacunarization and area of increased density).
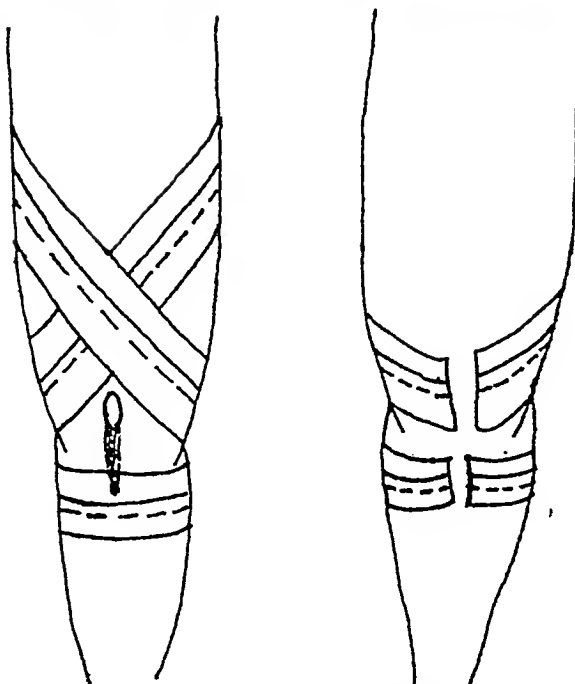

FIG. 5. Diagram of cross strapping.

and insofar as possible limit flexion of the knee. In addition, tape was applied in a circular fashion over the tubercle itself. In all cases we were careful not to encircle the extremity completely with tape.

COMMENT

It seems practically impossible to make any even relatively accurate estimate of the incidence of Osgood-Schlatter's disease in the general population, but the appearance of the thirteen cases of this series in nine weeks seemed to us an unexpected number. We have attempted an estimate (admittedly very rough) of the general incidence of this disease among the young men of the Center. All inductees arriving at the station were quartered in one division of the Center and reported to one dispensary for sick call. During the period of nine weeks covering this study approxi-

mately 1,024 men per week entered the Center which represents an incidence of 0.14 per cent of the general population of young males between the ages of seventeen and nineteen. It is well to remember, however, that this percentage might be somewhat higher at the age of puberty when the disease is reported to be seen most frequently. Certainly the cases reported in the literature are predominently at the age of puberty. Wilson, Fishell and Lagasa[5] report the incidence of Osgood-Schlatters disease to be 7 per cent of all knees examined in a New York hospital. To our knowledge, however, no one has attempted an estimate of the general incidence in the young male population and ours is at most an estimate.

SUMMARY

Thirteen cases of Osgood-Schlatter's disease seen in a period of nine weeks at a large Naval recruit training center are presented with a report of the x-ray findings and treatment, the latter was instituted while the patients were ambulatory and without restriction of their activity in any way. Forty-six and one-tenth per cent showed subjective improvement under anterior thigh taping as described, 15.4 per cent were not improved, 15.4 per cent were made worse and in 23.6 per cent we were unable to follow the patients. A roentgenographic classification is suggested and a rough estimate of the incidence of Osgood-Schlatter's disease among young males is submitted.

CONCLUSIONS

1. Osgood-Schlatter's disease may be precipitated and/or the symptoms aggravated by the increased exercise carried on during Naval training.

2. The doctor dealing with young men engaged in vigorous exercise should be conscious of the fact that in the young

military-age group the incidence of this disease may be greater than in general practice.

3. Military personnel may be definitely improved by conservative measures even if limitation of activity and complete limitation of the knee joint motion is impossible.

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TREATMENT OF CHRONIC ULCERS WITH CHLOROPHYLL*

REVIEW OF A SERIES OF FIFTY CASES

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THE large number of articles found in any one year's volume of medical literature on the treatment of chronic ulcers of varicose, diabetic, arteriosclerotic or other etiology, indicates that no one method of treatment has been found entirely satisfactory. Any addition to this mass of literature is, therefore, only justified if the series of cases treated is of significant size and the results obtained in that series are better than average.

Economically, chronic ulcers constitute an expensive medical problem. On the part of the patient they require frequent visits to the doctor over a prolonged period of time. Incapacity and absence from work is often a considerable item and the lesions often necessitate an outlay of considerable money in the purchase of dressings, bandages and various ointments. For the hospital such patients hinder rapid turnover because treatment is prolonged, and they place a burden on the nursing facilities because of the individual care which they demand.

Because of the inherent chronicity of ulcers, it has been the aim of all methods of treatment to effect healing as early as possible. Any new therapeutic preparation should contain agents which will increase the rate of healing. Many such substances have been proposed, yet their very number suggests that no one of them is uniformly successful.

Recently, as a result of the experimental work of several investigators,[1-6] the water-soluble derivatives of chlorophyll have been shown to possess tissue-stimulating properties. Smith and Livingston,[7] in a series of 1,372 experimental lesions produced in animals and treated with chlorophyll, showed healing time to be decreased by approximately 25 per cent in over two-thirds of their cases. These results together with other clinical reports concerning chlorophyll therapy were so impressive that it was thought worth while to treat a series of chronic ulcers with this material in an attempt to corroborate clinically their experimental findings. An historic review of our knowledge of chlorophyll in medicine was published by one of us (W.S.M.)[8] in July, 1946. Boehme[9] reported a series similar to the present study in April, 1946.

MATERIAL AND METHODS

From October, 1945 to August, 1946, fifty patients with chronic ulcers received topical treatment with a hydrophilic ointment containing water-soluble derivatives of chlorophyll. Of these, twenty-eight have been cases of varicose or stasis ulcers, six arteriosclerotic ulcers, six diabetic ulcers, five decubitus ulcers, two malignant ulcers and three ulcers of uncertain or undetermined etiology. Eighty-five per cent of the patients had been referred to the clinic because of intractable pain, failure of healing or for other reasons. In some the ulcers were a complication of a more primary disease: diabetes, congestive heart failure and spinal cord lesions.

The method has consisted in the local application of Chloresium ointment.* The ointment was applied in moderately generous quantities to a large square dressing which was then placed over the ulcer area and held in place with simple bandaging. Initially, dressings were changed every second day. Later the routine was modified and application of the ointment was made

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* The Chloresium ointment used in this investigation was generously provided by the Rystan Company, Inc., Mount Vernon, N. Y.

* From the Department of Medicine, Guthrie Clinic and Robert Packer Hospital, Sayre, Pa.

daily because it was found that dressings left unchanged for two days tended to become dry and it was thought that this decreased their therapeutic value. In the earlier cases with gross infection, 100,000 units of penicillin were added to a 1 pound jar of the Chloresium ointment. This was found adequate to control the markedly infected cases. Actually, however, most cases showing a moderate amount of purulent drainage cleared up so promptly with the chlorophyll ointment alone that during the latter two-thirds of the series no penicillin or other antibacterial agents were added to the Chloresium ointment. Smith's experimental findings appear to indicate that water-soluble chlorophyll itself is a mild antibiotic and probably increases tissue resistance to the action of bacteria.

In extremely painful lesions the dressings were changed several times daily during the first few days of treatment because it was found that by so doing the lesions became painless more quickly. Redressing more often than once daily was seldom necessary, however, because the soothing effect of a single application will usually last for twelve to eighteen hours. In attempting to evaluate the effectiveness of the chlorophyll preparation the other measures used in the treatment of ulcers were avoided with but few exceptions. Three patients in the series with varicose ulcers had saphenous vein ligation and this accepted surgical procedure was recommended subsequently to all suitable patients with varicose veins. We abstained conscientiously from the use of pressure bandages, radiant heat, boots, etc., to avoid obscuring the results. When the general condition of the patient permitted, he was given ambulatory privileges and 30 per cent of the cases in this series were treated as out-patients, pursuing their more or less usual activities and occupations during treatment.

RESULTS

We have been gratified and encouraged by the results that we have observed in this series of cases. (Table 1.) As has been mentioned approximately 85 per cent of the patients had been referred to the clinic by general practitioners, over a radius of about 150 miles, because they had constituted long, chronic and refractory prob-

TABLE 1

Etiology	No. of Cases	Im-proved*	Unin-proved*	Healed†
Varicose and stasis...	28	28	0	20
Arteriosclerotic......	6	5	1	3
Diabetic...........	6	5	1	3
Decubitus..........	5	4	1	2
Malignant..........	2	2‡	0	0
Undetermined.......	3	3	0	2
	50	47	3	30

* Based on a period of observation varying from one to four months.
† Based upon follow-up examinations or communications of forty of the patients two to seven months after treatment.
‡ The term "improved" in the case of malignant ulcers indicates a decrease in the local discomfort, odor, infection and induration; not, of course, decrease in the malignant process.

lems in therapy. A few others had been treated with inadequate success over a long period of time by our own out-patient department. Most of the patients had received a variety of therapeutic measures, including prolonged periods of bed rest in a number of instances.

It appears to us that in general, patients with the greatest degree of vascular insufficiency showed the poorest response to chlorophyll therapy as might well have been anticipated. Healing time was almost in direct proportion to the degree of vascular involvement. Diabetic ulcers, in patients showing a considerable amount of arteriosclerosis, took longer to heal than those having only one or the other of these conditions. In one patient having syphilis, diabetes and arteriosclerosis no acceleration of the healing time was noted. Yet even those patients with maximal involvement of the vascular tree and whose lesions had been stationary or progressive for months

or years showed progressive healing although complete closure of the ulcer was less prompt than in those with better preservation of their peripheral circulation. The varicose and stasis ulcers responded best of all and in some of these the effect of chlorophyll was often quite remarkable.

It has been our observation in this series of chronic ulcers that water-soluble chlorophyll therapy satisfies two of the primary requirements of the "ideal" substance for the treatment of this condition: It relieves the subjective symptoms which are secondary to the ulcer and it promotes tissue growth and healing in the majority of cases. In persons who complained of pain from the ulcer this was quickly alleviated by application of the chlorophyll ointment. In a few dramatic instances patients volunteered the information that for the first time in weeks or months they were comfortable and slept soundly during the night following their admission and the application of the ointment. As a rule pain was no longer a problem after several days' treatment and sedation could be discontinued after the first twenty-four or forty-eight hours. No untoward local reactions were experienced although one patient complained of an unexplained burning pain at the ulcer site for a few minutes after the first application of the ointment. The relief of such associated symptoms as pain, itching and burning due to stasis dermatitis, burns, wounds and a variety of other conditions has been mentioned in separate reports by Langley and Morgan[10] and Morgan.[11]

The objective evidence of healing was apparent in every patient except three, two of them having malignant ulcers; the criterion for healing is an appreciable and measurable decrease in the size of the ulcer. It was uniformly noticed that shortly after therapy was begun the tissues took on a healthier appearance and new granulations were visible. The inflammation and tenderness was diminished as a rule and often completely disappeared. Ulcers which were secondarily infected and foul-smelling were relieved promptly of both the discharge and odor after application of the ointment for a day or two. When there was an accompanying stasis dermatitis, this uniformly improved.

ANALYSES

Twenty-eight cases of varicose and stasis ulcers were treated. All of the patients showed improvement and twenty of twenty-four patients who could be followed for two to seven months remained healed. Saphenous vein ligation was performed in only three subjects although it was recommended in all suitable cases.

Six patients with ulcer of the lower extremities of arteriosclerotic etiology received similar treatment. Only one patient in this group did not show improvement. This was a sixty-five year old male who had had an amputation of the left leg several years before because of arteriosclerotic gangrene. He was re-admitted because of gangrene of the right great toe. Débridement of the necrotic tissue and application of chlorophyll ointment for about one month, together with the usual supportive measures, failed to produce appreciable improvement and amputation of the toe was performed. A somewhat similar case (Case II) did show a good response and complete healing occurred.

Six diabetic ulcers were treated with Chloresium ointment. Three of these healed promptly. One patient with associated syphilis and advanced arteriosclerosis showed very slow healing. One showed progressive improvement but incomplete healing, presumably because of an associated osteomyelitis. The sixth patient showed no benefit from an intensive two-week's trial. This patient was a diabetic of twelve years' duration with an ulcer for two years. Roentgenograms showed marked arteriosclerosis of the arteries of the feet. Diabetes had been controlled elsewhere with no effect on the ulcer. It was suspected, although not proven, that this patient's ulcer was complicated by the presence of an inadequately draining sinus. (Case III.)

Five patients with decubitus ulcer were treated. These are the most difficult of all ulcers to treat and the results achieved with the chlorophyll ointment were satisfactory but not spectacular. All except one of the patients improved both subjectively and objectively as shown by progressive decrease in the size of the lesions. Only two patients showed complete healing and one case, a woman forty-four years old with an inoperable spinal cord tumor who had both urinary and fecal incontinence, was considered a failure. This patient died of urinary tract and pulmonary sepsis. It was observed, however, that water-soluble chlorophyll served to alleviate pain and produced a much healthier ulcer than any therapy previously employed; granulation tissue formation with healing occurred although at a much slower rate than was noted in ulcers of other etiology.

Two malignant ulcers were treated, one before and one after the diagnosis of carcinoma was established. The initial biopsy from the first case reported only chronic inflammatory tissue. The chlorophyll ointment produced subjective benefit and seemed to reduce the amount of inflammatory reaction around the ulcer. A second biopsy showed basal cell carcinoma of the cheek and the patient was treated with roentgen therapy. The second patient had ulcerative squamous cell epithelioma of the left knee, confirmed by biopsy, on whom several skin grafts had been applied at another hospital. This patient's lesion was extensively infected and extremely foul-smelling. The application of chlorophyll ointment rapidly eliminated the odor, cleaned up the infection and produced new granulations in the base of the ulcer. X-ray therapy was then given but a recurrence developed for which mid-thigh amputation was required.

Three ulcers in which an etiologic diagnosis remained in doubt received chlorophyll therapy. The first was a man sixty-five years of age with no apparent arterial or venous insufficiency of the extremities who had had repeated admissions because of recurrent indolent ulcers of the right lower leg. The lesions had a surrounding dermatitis and a similar dermatitis of the thighs and arms. A great variety of therapeutic agents had been used, including irradiation. An equally extensive variety of diagnoses had been applied, including lichen planus, lichen simplex chronicus, lichen vidal, dermatitis factitia and dermatitis venenata. The patient was treated for twenty-one consecutive days with chlorophyll ointment. He was discharged with the indolent ulcer practically healed and was given a supply of the ointment for home applications. A subsequent letter dated nine months later stated that the ulcer had remained healed and quiescent. This was the longest remission that had been recorded since his first appearance at the clinic three years previously.

The second of these ulcers of undetermined etiology occurred in a woman forty years old who for twenty years had had many ulcers of the left lower leg which had healed and had repeatedly broken down. She has been repeatedly treated at the Robert Packer Hospital since July, 1944 with Unna's boots, red cell paste, elastic bandages, etc., with temporary improvement. Varicose veins were considered the cause of the ulcers but were never proven and none were apparent. Her most recent admission was from March 6th to April 21, 1946. On this admission the patient had an ulcer which almost encircled the left leg above the ankle and which had been present for eight months. Six weeks of treatment with chlorophyll ointment caused rapid decrease in the size of the lesion by about 50 per cent but thereafter healing became extremely slow. It was believed that the ulcer was probably ischemic in nature although the exact etiology was unknown. Serologic tests for syphilis were negative in this and in the other two patients of this group with uncertain etiology. Other laboratory studies were likewise non-contributory.

The third of these indolent ulcers of undetermined etiology was observed in a man

of forty-three years who for five years had had recurrent superficial ulcerations of both ankles. When admitted to the hospital, he had an open ulcer over the internal malleolus of each leg. That on the right measured 4 by 10 cm. and had been present for two years. The one on the left measured 2.5 by 2.5 cm. and had recurred following a bruise of the left ankle three months before. Local and general physical examination were negative. The lesions had been treated for some months in the out-patient department with gentian violet and other local applications but without significant results. The patient was hospitalized for five days' therapy and continued treatment as an out-patient. On an ambulatory status both ulcers gradually improved and when last seen, eight weeks following admission, the ulcer on the left leg measured 1.5 by 1.0 cm. and that on the right was 2 by 2 cm. Both had become asymptomatic and had shown progressive diminution in size in spite of the fact that the patient has continued to work forty hours weekly as a bench worker in a machine shop.

CASE REPORTS

CASE I.   A sixty-nine year old white female was admitted to the hospital with arteriosclerotic heart disease and congestive heart failure. Examination disclosed the additional presence of a painful chronic ulcer of the left leg. The patient stated that the ulcer had been present for the past thirty-four years during which time it had never healed. It had occurred spontaneously following a "milk leg" which appeared subsequent to childbirth. Healing had failed in spite of repeated efforts by various methods and by physicians since the time of its onset, and during this time there had been several periods of complete bed rest. Examination of the left lower extremity revealed a small ulcer measuring 2 by 2 by 0.5 cm. on the superior portion of the internal malleolus. The base of the ulcer was grayish in color, unhealthy in appearance, although not frankly infected, and was quite tender to touch. The ulcer was surrounded by an area of inflammation and by a large zone of chronic scarring and hyperpigmentation. Numerous varicose veins were present in both legs. The arterial pulsations were normal.

The ulcer was dressed daily with chlorophyll-penicillin ointment. Progressive healing was noted from the start of treatment. After several days the surrounding inflammation had disappeared and much of the tenderness was relieved. Eight days after treatment had begun the ulcer measured about 0.5 cm. in diameter and six days later it was completely healed. Inasmuch as the danger of recurrence of the ulcer was so obvious, it was recommended that saphenous ligation be performed. This was done after healing of the ulcer was complete. The patient was discharged from the hospital two days later. Follow-up examination four months later indicated no signs of recurrence of the ulcer. (Fig. 1.)

Comment.   The case of a patient with a thirty-four-year old ulcer which healed in fourteen days is described in some detail largely because of the outstanding result. It was the best result in our series of twenty-eight stasis ulcers although several others approached this in their rate and manner of healing. One varicose ulcer of three months' duration healed in eight days. Two others healed in nine days whereas one required four months. In a condition with so many variables of severity, age and associated conditions an "average" loses its meaning and statistical importance but, as the series of cases increased, we formed the clinical impression that chlorophyll ointment produced better and quicker results than measures previously used.

CASE II.   A seventy-four year old farmer was admitted to the hospital on October 23, 1945, because of arteriosclerotic gangrene of the right great toe. About three months earlier the patient noted discoloration of the nail and base of the toe. This became worse in appearance, progressively more painful and interfered with the patient's sleep. The toenail was removed by his physician at home and ointments were applied without relief of pain or change in appearance. For two weeks prior to admission there had been considerable swelling of the ankle and foot.

Physical examination showed a well pre-
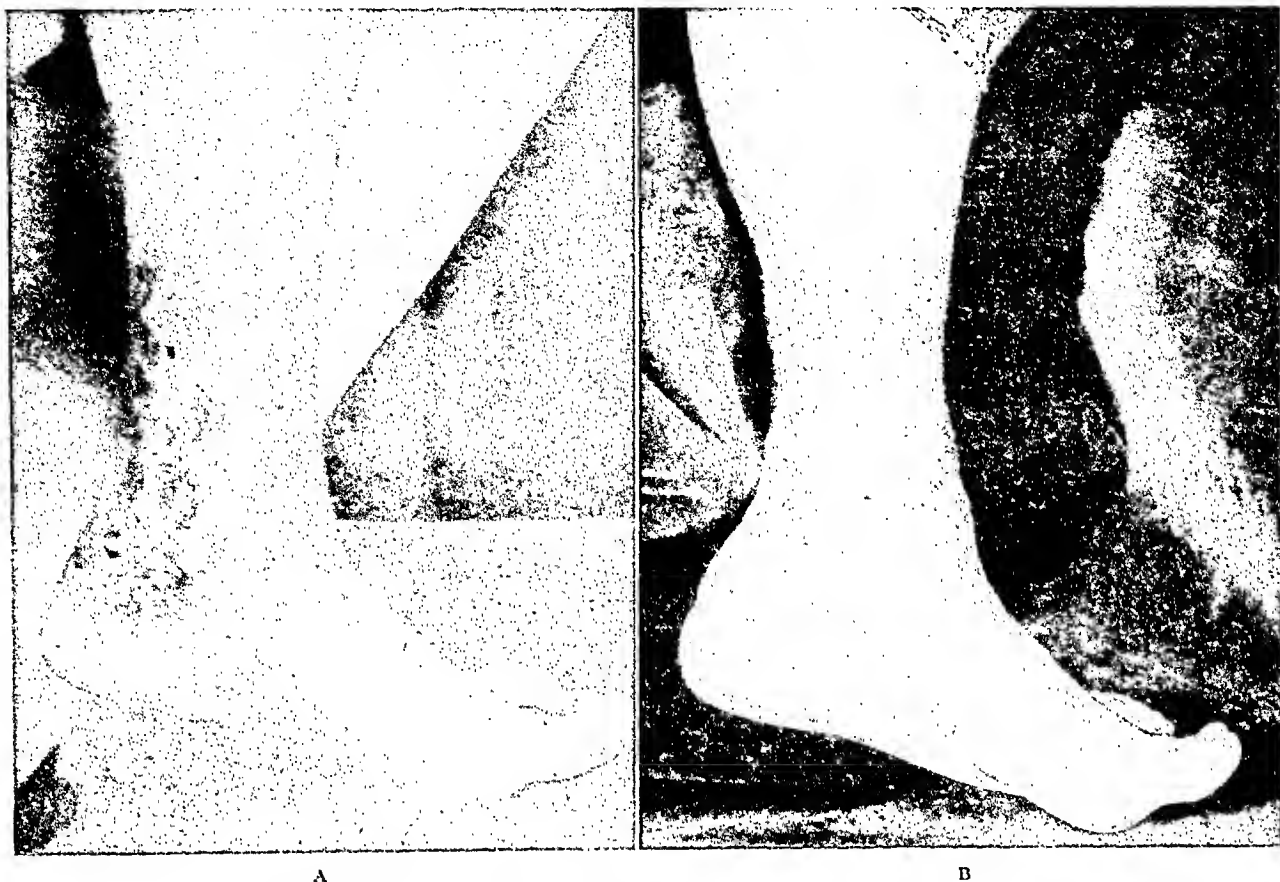
A   B

FIG. 1. A and B, showing the degree of healing of the ulcer in Case 1 over a two-week period.

served, elderly white male who did not appear acutely ill but obviously was in considerable pain. Blood pressure was 140 systolic, 85 diastolic, the pulse rate was 76 and the temperature was 98.6°F. Positive findings were limited to the extremities. Dorsalis pedis and posterior tibial pulsations were absent in both feet. The right foot showed a large, black slough covering the surface of the great toe; the nail was gone. A line of demarcation was present near the base of the toe and there was considerable inflammation of the adjacent area of the foot. Although the pulses were absent, the feet were not strikingly cold and there were good popliteal and femoral pulses. Sclerosis of the radial arteries was marked.

The patient was given bed rest, vasodilating agents, sedatives and Buerger's exercises. The slough was softened with warm saline soaks and part of this dead tissue was removed seven days after admission. Chlorophyll ointment was applied thereafter every second day, and by the end of ten additional days the pain had decreased, the color was markedly improved and purulent discharge had ceased. The patient was discharged from the hospital on November 10th

and given a supply of chlorophyll ointment and dressings for treatment at home.

He was readmitted eighteen days later. The foot was of good color but there was an area of hard eschar on the dorsum of the toe which it seemed desirable to remove. The eschar was softened with Chloresium solution for three days and then was excised. Thereafter, chlorophyll ointment was applied every second day and after six days the patient was comfortable and able to walk to the bathroom. The ulcerated area was granulating and had a healthy pink color. Adjacent inflammation was absent. Granulation and healing progressed and on December 22nd the patient was discharged, twenty-four days after his second admission. He continued to be ambulatory at home, applying chlorophyll ointment daily, and returned to the clinic at weekly intervals. By January 19, 1946, the toe had completely healed and the patient was comfortable and free from pain. He was advised regarding protection of the foot from cold and trauma. A letter from the patient dated June 3, 1946, stated that the toe had remained healed and that he had resumed part of his farm work,
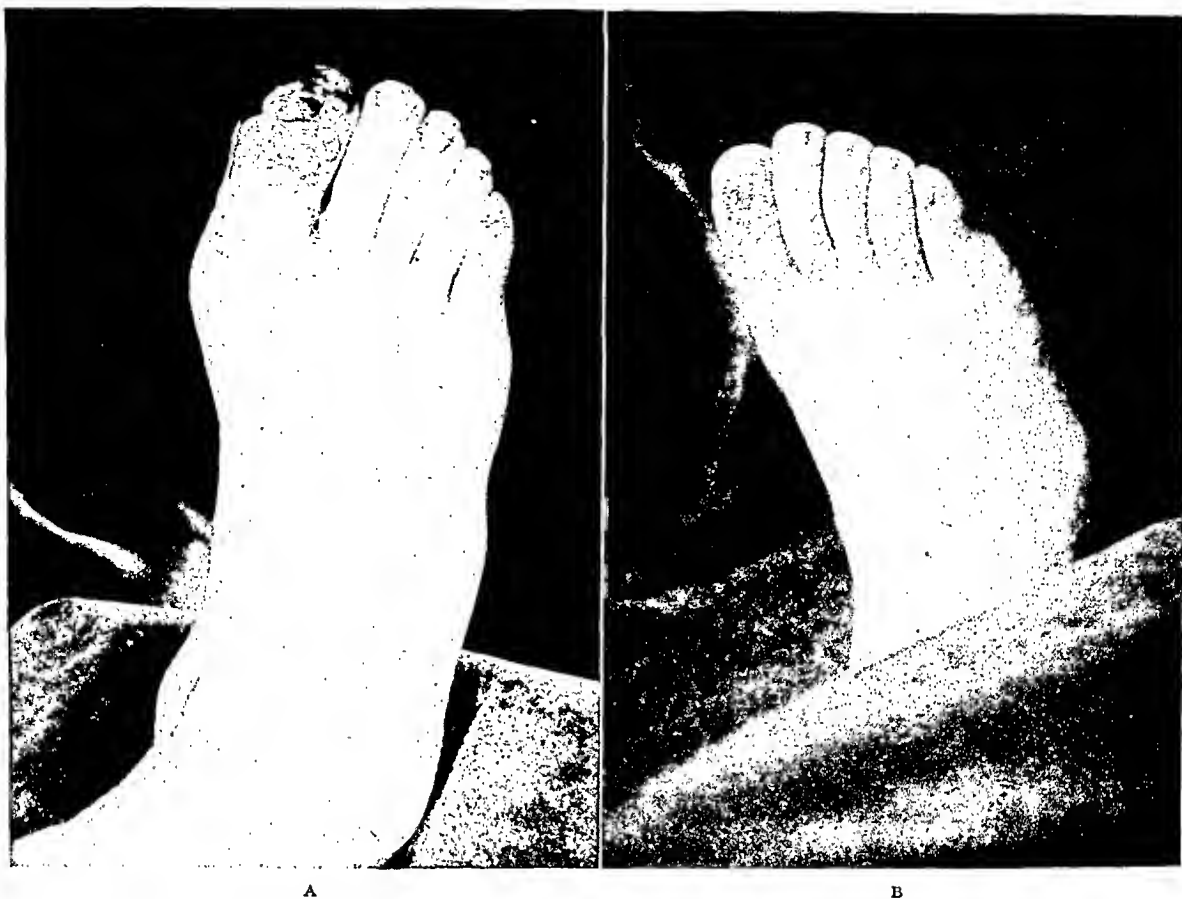
FIG. 2. A and B, an arteriosclerotic lesion of the right great toe treated for nine weeks with chlorophyll ointment, showing the degree of healing (Case II).

doing the chores and driving the tractor. (Fig. 2.)

Comment. Although healing in this case required eighty-eight days of observation and treatment, we believe that conservative therapy was warranted since the patient ultimately had a useful and comfortable extremity. After the slough and eschar had been removed, so that the ointment could contact more viable tissue, we gained the impression that chlorophyll added appreciably to the rate of healing and the alleviation of pain.

CASE III. A sixty-three year old white, diabetic female was admitted to the hospital April 26, 1946, because of a painful ulcer near the base of the right great toe which had not responded to treatment by her local physician. The patient had had diabetes for six years and the ulcer had appeared about four months before admission, following slight trauma. Her initial blood sugar was 246 mg. per cent. The diabetes was controlled with diet and protamine zinc insulin. The ulcer was treated for two weeks with plain sterile dressings following excision of a small area of necrotic tissue. A roentgenogram showed probable osteomyelitis of the terminal phalanx. No change in the size of the ulcer occurred and it remained painful. Daily dressings were begun, using chlorophyll ointment. The lesion became more comfortable and healing began. The ulcer was entirely healed in ten days and dressings were discontinued. The patient was seen fifteen months after discharge from the hospital and there had been no recurrence of the ulcer. She remained active in her housework and was comfortable. (Fig. 3.)

Comment. Bed rest and control of the diabetes may have played a part in the healing of the ulcer. Nevertheless, healing was not apparent until the therapeutic regimen included the daily application of chlorophyll ointment, and it appeared to
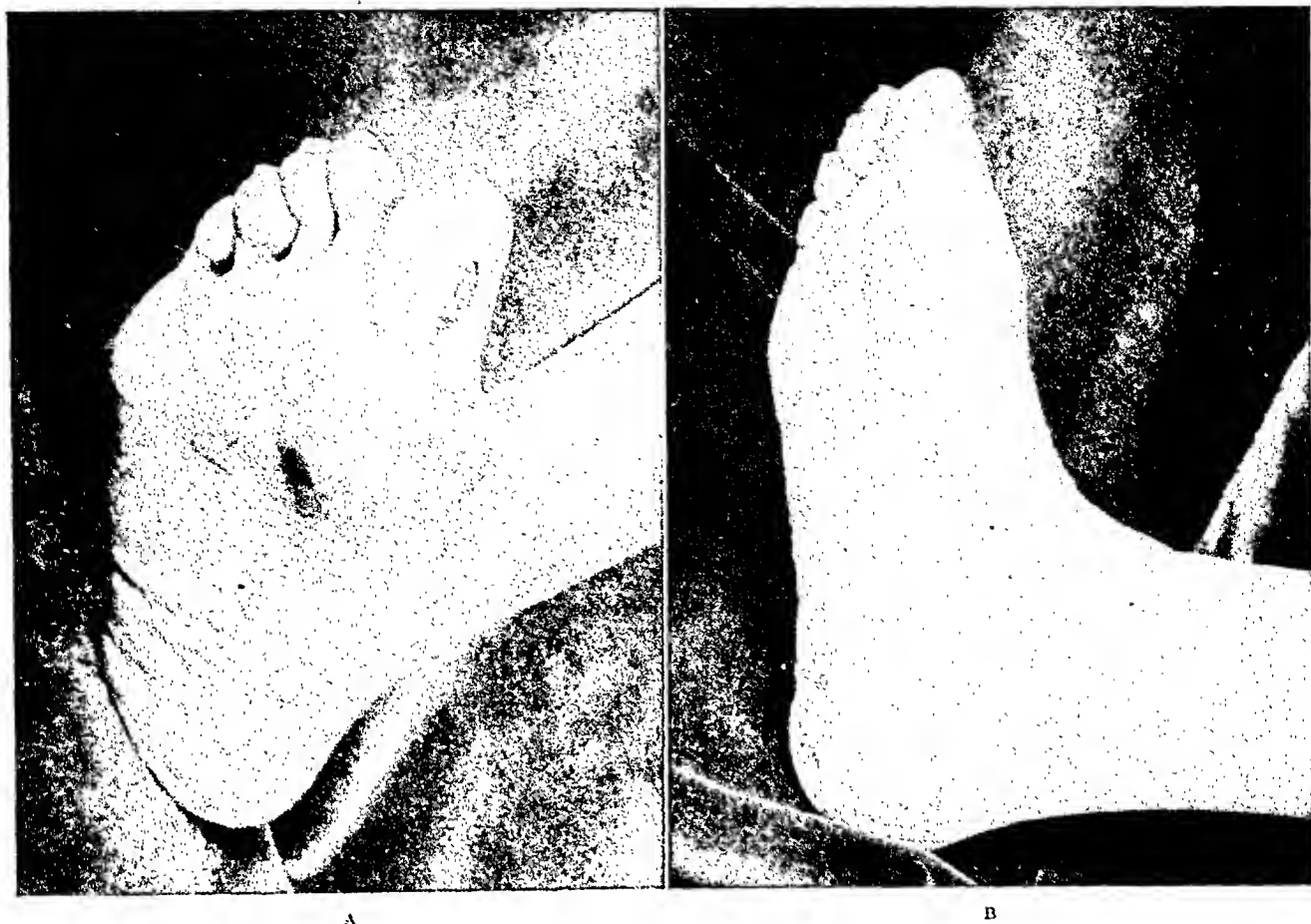
A   B

FIG. 3. A and B, results of treatment of a diabetic ulcer of the right great toe over a period of ten days with local application of chlorophyll ointment (Case III).

us that this agent contributed to the rapid healing.

SUMMARY

Gratifying results have followed the application of an ointment containing the water-soluble derivatives of chlorophyll (Chloresium) in a hydrophilic base in fifty indolent ulcers.

Subjective relief of symptoms was expressed by every patient during the course of treatment.

Objective evidence of healing was noted to an appreciable degree in all except three patients.

We believe that chlorophyll therapy should have a place in the management of chronic ulcers although it cannot be used to the exclusion of other recognized methods of medical and surgical management.

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GASTRIC DIVERTICULA*

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THE presence of diverticula in the gastrointestinal tract does not elicit much enthusiasm from a surgical viewpoint. However, when a gastric diverticulum is demonstrated, the rarity of this lesion captivates the interest of all. Literary studies definitely categorize gastric diverticula among the rarities in surgery. This entity has a reported incidence ranging from 0.001 per cent to 0.5 per cent. These figures are based upon roentgenologic and gastroscopic studies as well as surgical and necropsy specimens.[1,7,8,9,10] Thus it is an unusual experience to encounter many of these diverticula during the professional life of any one surgeon.

Gastric diverticula are sac-like evaginations of the wall. The cavity is continuous with the stomach by an orifice or neck-like formation. Basically diverticula may be congenital or acquired. The walls of congenital diverticula are full thickness corresponding to those of the stomach. In the acquired type, diverticula may be lacking in some gastric layers. Diverticula may be classified as follows: (1) congenital diverticula, (2) acquired diverticula and (3) pseudodiverticula.

Etiology. Congenital diverticula find their origin in embryonic life. The exact mechanism of origin is questionable. According to some authors an atavistic phenomenon associated with pancreatic development has been incriminated.[2] Others believe that diverticula near the pylorous especially are due to dispersed pancreatic anlages.[1] Suffice it to state that congenital diverticula result from malformation or disturbance in development. All gastric layers are present. Congenital diverticula are round, smooth cul-de-sacs, usually with a wide neck.

Acquired diverticula offer opportunity for speculation as to their origin. The walls of acquired diverticula consist of a mucosa, submucosa, varying numbers of muscular fibers and a serosa. They may be derived from hernial formations of the mucosa across vascular orifices.[4] The passage of the mucosa through existing vascular orifices in a weakened wall may be caused by an increased intragastric pressure during the process of digestion. Another etiologic factor may be a circumscribed paresis of the gastric wall.[2] This paresis is assumed to result from a chronic neuritis of a branch of the vagus nerve. Factors closely resembling those producing an esophageal diverticulum may be considered. Especially is this true in diverticula near the cardia. At this site the longitudinal muscles of the stomach divide into two muscular fasciculi. Within the region of the division the muscularis is formed only of circular muscle fibers.[10] This is a weakened area which is the basis for the development of a diverticulum. This class of diverticula have narrow orifices.

Pseudodiverticular formations may be formed by pathologic conditions producing a pouching of the stomach walls. The most common of these causes are perigastric adhesions, ulcers and other inflammatory lesions. (Fig. 1.)

Gastric diverticula are usually seen in men. The location of choice apparently is the lesser curvature, at the cardiac end of the posterior wall. However, there is no reason to doubt the possibility that a diverticulum may occur elsewhere. Since the localization at the cardiac orifice is most common, at times it has been termed the "cardiac orifice diverticulum."

Pathology. Gastric diverticula may assume various shapes and sizes. The orifices may be large or small with or without a pedicle simulating a neck-like appearance. This pedicle enters the diverticulum. The

diverticulum itself is a pouch-like projection. The diameter of the sac may range from 1 by 2 cm. to 5 by 6 cm.[5] The diverticulum is lined with gastric mucosa. Its wall, however, is thinner than normal stomach.

Histologic section demonstrates gastric diverticula to consist of mucosa, submucosa, muscle fibers and serosa. The wall, however, is composed of compressed glandular elements with flattened epithelial cells which may be only one layer thick. Beneath these cells is seen a layer of lymphoid tissue. The muscular elements are an attenuated layer of poorly developed muscle bundles. Outside this may be seen a little connective tissue and a serosal covering.

Associated gastric disease has been reported in proven cases of diverticula.[5,8,9] The combination of diverticulum and ulcer has been stressed; however, many diverticula are not associated with ulceration. Consequently, while the symptoms of uncomplicated gastric diverticula are often suggestive of peptic ulcer, it must not be forgotten that an ulcer may be associated with a diverticulum.[5] Neoplasms have been encountered in diverticula. Adenomyoma, fibrosarcoma, sarcoma, cancer and precancerous lesions have been recorded as present in gastric diverticula.[9] This underlying condition often causes the symptoms which are attributed to the diverticulum. Thus the true nature of the lesion is determined only at operation or necropsy.

Clinical Picture. A definite clinical pattern cannot be postulated for gastric diverticulum. In truth, many patients with uncomplicated diverticula are asymptomatic. When symptoms do occur, they are not manifested until the fourth or fifth decade of life. A logical assumption is that diverticula with narrow necks are more likely to cause symptoms because of difficulties in emptying. Associated disease in diverticula or pseudodiverticula usually produce clinical symptoms.

Because this lesion is located in the stomach, the manifestations of a diverticulum may produce any symptom generally associated with gastric disturbance. The most common symptoms are: (1) Pain, epigastric or upper abdominal; dull aching or burning in type; may or may not be relieved by food; (2) vomiting; (3) belching; (4) epigastric tenderness, slight to moderate; (5) dysphagia when lesion is large and near esophagus; (6) bleeding; hematochezia, hematemesis are rarely present, but may occur; and (7) the sensation of pressure and a feeling of gastric distention associated with hematemesis has been reported.[1]

In summation, the clinical picture depends upon the site and size of the diverticulum and the presence of ulceration or food retention within the sac. The usual biochemical studies including gastric analysis rarely assist in the diagnosis of uncomplicated gastric diverticulum.

Diagnosis. Since the clinical picture is not characteristic, the roentgenologist often makes the diagnosis. Even he may find difficulty in interpreting the finding. The diverticulum may be missed if studies are made only in the postero-anterior position. Because of this fact studies in the erect, prone, oblique and lateral positions have been recommended.[1] Hence it can be appreciated that the diagnosis of a true gastric diverticulum is impossible without the aid of x-ray. At times roentgenologic diagnosis is extremely difficult. For this reason the following three postulates must be fulfilled in arriving at a roentgenologic diagnosis of gastric diverticulum:[7] (1) The sac must be mobile and unattached to extragastric structures; (2) the shadow must be well defined, smooth, regular and demonstrated from various angles; (3) usually no tenderness is elicited over the area of filling.

Gastroscopy has limited value as a diagnostic aid because of the location of diverticula in an area which cannot be visualized. When the lesion can be seen with a gastroscope, it closely resembles a bladder diverticulum. It has a round, sharp orifice; the diameter of the opening is
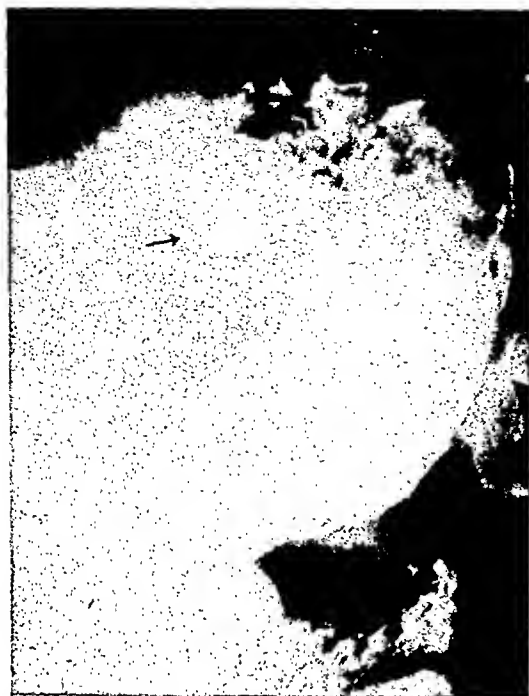
FIG. 1. Roentgenogram of a pseudodiverticulum in a fifty-five year old man with a perforating gastric ulcer found at operation.

smaller than the greatest diameter of the diverticulum. If light can be reflected into the opening, it is seen to be covered with a smooth red mucosa. Normal rugae are discerned.[7]

Differential Diagnosis. Before concluding that a lesion is a diverticulum, other diseases in which destruction of the stomach wall occurs must be eliminated. The most confusing lesion to be differentiated is a penetrating gastric ulcer,—often called Haudek's niche. (Fig. 1.) This may be eliminated by the usual location of the diverticulum, the absence of contralateral spasm, infiltration and rigidity. Moreover the normal mucosal pattern can be demonstrated in a diverticulum but cannot in a gastric ulcer. The presence of an ulcer history and gastric analysis are valuable adjuncts in the differential diagnosis.

Diaphragmatic hernia occasionally is confused with a gastric diverticulum. Adequate x-ray studies will demonstrate the presence of a diaphragmatic hernia. The visualization of a gastric shadow in the chest with a gas bubble and fluid level is almost pathognomonic of diaphragmatic hernia.

Very often the diagnosis of gastric ulcer or carcinoma is made only at the time of operation or autopsy, even as the presence of a diverticulum is only revealed by these

TABLE I

COMPOSITE CHART OF THREE CASES DISCUSSED

Case	Age	Sex	Gastric Symptoms	Cause of Death
I	52	M	None	Acute coronary occlusion
II	45	M	From Family { 1. Epigastric pain 2. Nausea 3. Questionable hematemesis	Accidental death; brain laceration
III	62	M	None	Congestive heart failure

procedures. Our own experience with three cases of gastric diverticulum falls into this category.

CASE REPORTS

We have seen many instances of diverticula demonstrated by x-ray. At operation the underlying lesion was an ulcer forming a pseudodiverticulum. However, three cases of true uncomplicated gastric diverticula were seen at autopsy in adult men who died of causes not related to the gastric findings. Table 1 and Figure 2 illustrate the salient features of these cases as well as a diagrammatic picture of the diverticula.

These three cases presented features which comply with the pattern of simple, uncomplicated gastric diverticula as previously discussed.

Complications. Subsequent to the accumulation of undigested food in a diverticulum, decomposition occurs which is a factor in the development of inflammatory changes. Edema of the mucosa accompanying the inflammation may produce stenosis of the diverticular neck. Thus a vicious cycle is established which further increases fermentation and accompanying inflammation. The development of com-

plications may be explained in this way. Hence gangrene and inflammation of the serosal surface may occur. This may lead to erosion of a blood vessel and fatal hemorrhage. Diverticula must be considered as a cause for idopathic sepsis.

is the high incidence of associated disease in diverticula.

In the presence of active gastric symptoms, surgery is necessary when, after an adequate medical regimen, symptoms persist. Diet may alleviate symptoms somewhat but
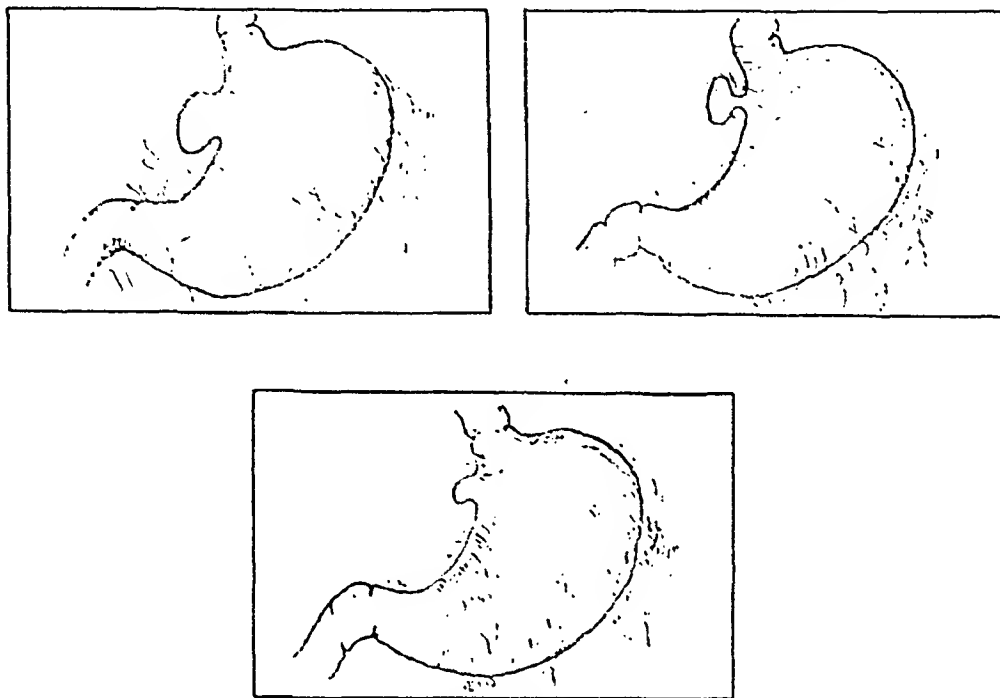

FIG. 2. Cases I, II and III.

A high incidence of associated disease has been reported in gastric diverticula. Benign and malignant tumors have been recorded such as leiomyosarcoma, adenomyoma, fibrosarcoma, sarcoma, carcinoma, adenoma and myoma.[7] Recurrent slight bleeding and massive hemorrhage have occurred. In addition to blood vessel erosion, bleeding has been attributed to stagnation of food particles, trauma from harsh food or the retention of a highly acid gastric content. Bleeding from a gastric ulcer contained within a diverticulum is often the first sign of a diverticulum.[7] Thus the combination of diverticulum and ulcer has been stressed.[5]

Treatment. Surgical treatment is indicated for gastric diverticulum because of the inherent tendency of irritative lesions of the stomach to become malignant. Whenever a suspicious lesion simulates cancer, surgery is imperative. Another factor stimulating surgical intervention

rarely effects a cure. Usually patients are not relieved at all by a dietary regimen. When food is retained in the sac, medical treatment is ineffectual. Ulceration, bleeding and perforation have occurred while patients were on palliative treatment.[8] Some authors have advocated the surgical removal of gastric diverticula if symptoms are present.[7] The choice of treatment must be a matter of judgment, with surgical intervention advocated if the occasion demands it.

Surgical treatment consists in dissecting and liberating the sac similar to the procedure for esophageal diverticulum. The neck is then crushed, ligated and the operative site inverted.

Prognosis. Asymptomatic gastric diverticula without surgical treatment must be considered as having the same prognosis as bladder or esophageal diverticula. When symptoms occur the prognosis is altered because of the possibility of associated

diseases, especially ulcer and neoplasm. In the presence of these pathologic entities, the prognosis is the same as that for gastric ulcer and neoplasm. A diverticulum containing either ulcer or cancer may produce the same complications as these lesions without a diverticulum.

Surgical treatment of uncomplicated gastric diverticula alleviates the symptoms caused by it. Usually the mortality is very low; no serious sequelae or recurrences are anticipated following surgery.[3] When gastric resection is necessary for ulcer or malignancy, the prognosis is the same as for these lesions without a diverticulum.

SUMMARY AND CONCLUSIONS

1. The presence of gastric diverticula, according to the literature, must be listed as a rarity in surgery.

2. Etiologically this lesion may be congenital or acquired. Pseudodiverticula may be formed which are difficult to differentiate from true diverticula.

3. No definite clinical picture categorizes this entity. Diagnostic aids are x-ray and gastroscopy. Often asymptomatic diverticula are found at operation or autopsy.

4. The high incidence of associated benign and malignant conditions is stressed.

5. Treatment is based on judgment. However, in the presence of indecision, surgical intervention should not be denied the patient with symptoms.

6. Surgery is advocated because of the high incidence of associated lesions in gastric diverticula.

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INCISIONAL HERNIA REPAIRED WITH TANTALUM GAUZE*

PRELIMINARY REPORT

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ALTHOUGH a number of reports of successful repair of skull defects by tantalum plates have appeared in the literature,[1-3] we can find no references to the surgical employment of this metal in the form of gauze. It therefore seems justifiable to call attention to its value and to report a case in which the tantalum mesh was used with satisfaction.

Tantalum, the seventy-third element in the periodic table, was discovered by Ekeberg[4] in 1802, and was so named because he found it to be "tantalizing" when he attempted to dissolve it in mineral acids. It is manufactured in the form of sheet, foil, ribbon, wire, screws, plates and gauze or screen. Among its physical properties are its strength and malleability. It is ductile and may be drawn into fine wire which appears to be an excellent suture material. Burke,[5] in 1940, used it experimentally in animals and found it biologically inert. It was probably first used in human surgery by Fulcher[1] who employed it for the repair of skull defects. Later investigators have confirmed its value.[2-4] McNealy and Glassman[6] have recently reported satisfactory results with perforated Vitallium plates in the repair of hernia. Whether or not tantalum gauze will prove superior to the latter for this purpose remains to be seen, but its physical characteristics and the ease of its employment give promise that it may.

No surgeon prefers to substitute foreign material for human tissue, yet there definitely comes a time in the realm of reconstructive surgery when autogenous tissue does not seem to be the best answer, at least in the quantities desired and needed. The neurosurgeon has perhaps realized this more in his quest for ideal material in the repair of skull defects. Orthopedic surgeons are now finding less need for foreign substances as more is known about the advantages of bone grafts in replacing the use of metal plates. Generally speaking, plastic surgeons refrain from the use of foreign material. Perhaps this is due in part to the fact that their reconstructive needs usually are not great and that their work is most often located in parts of the body which do not lend themselves to foreign body substitution.

At one time the question as to whether or not incisional hernias belonged to the plastic or general surgeon was a matter of controversy. It is now generally conceded that such hernias belong within the realm of general surgery. However, in order to repair such defects the methods of plastic or reconstructive surgery must be employed.

No surgeon contemplates the repair of a massive incisional hernia with great enthusiasm, especially in the upper quadrants. The difficulty, and at times the impossibility of bringing together the supportive fascial tissues without tension or even with tension, puts such cases in the category of problem surgery. Various expedients have been tried to overcome the difficulty. Among these are fascial transplants, pedicled fascial flaps and cutis grafts. Since, as remarked by McNealy, the transplanted fascial flaps and folded fascial flaps are often no stronger than that at the hernial

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FIG. 1. Front view of an upper abdominal incisional hernia which resulted from emergency surgery for a ruptured peptic ulcer one year previously.

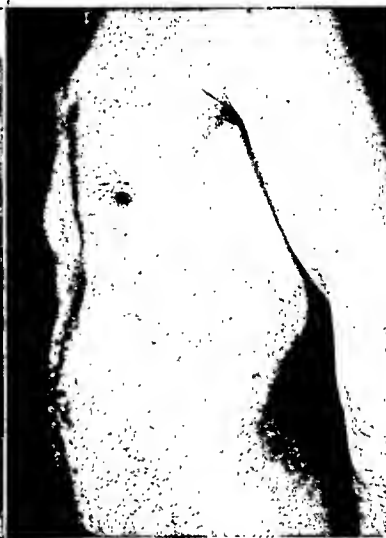FIG. 2. Lateral view of the same patient as Figure 1.

FIG. 3. Four-week postoperative view after tantalum implantation. The hernia is obliterated.

FIG. 4. Lateral postoperative view four weeks after implantation of tantalum.

site, repair attempted with the use of this material may be foredoomed to failure. Cutis grafts have not proved generally successful. Moreover, they have the disadvantage of leaving unsightly scars at the donor site, a matter of considerable importance in certain classes of patients. Any substitute which provides solid support without undue complications is therefore welcome.

It would appear that tantalum screen or gauze at this time most nearly approaches the ideal in selected cases.[7] Tantalum gauze is not yet available for general distribution.* "To use this material in herniorrhaphy the secret seems to lie in the fact that a repair can be done without

* We were able to secure the material and a discussion of its use from the Ethicon Suture Laboratories, Division of Johnson & Johnson.
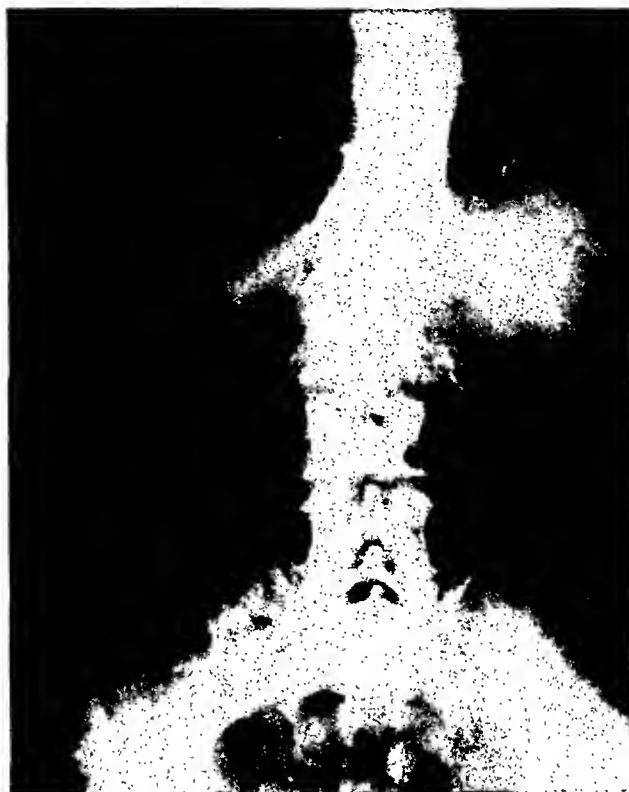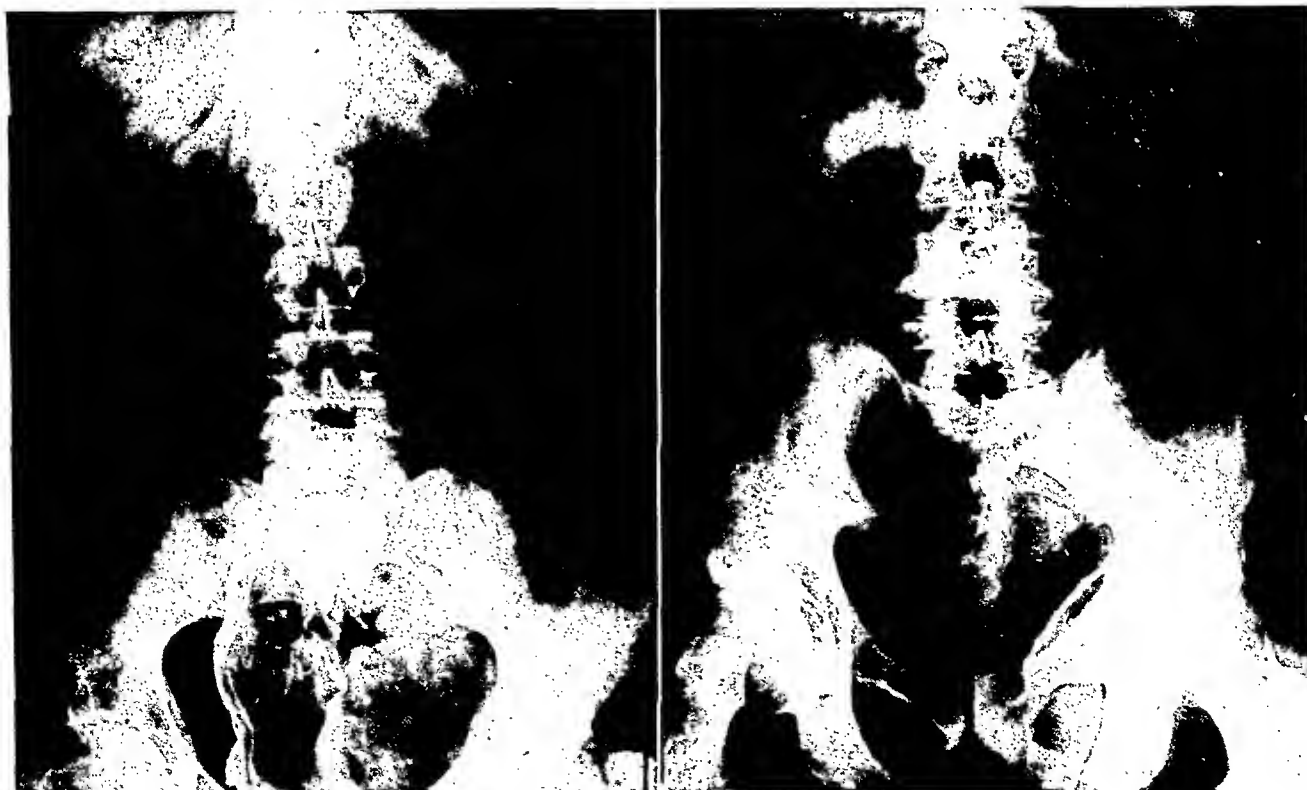
FIG. 5. X-ray shows tantalum in place in upper abdomen.

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FIG. 6. Lower midline incisional hernia following cesarean section. X-ray shows tantalum in place in lower midabdomen.

FIG. 7. Tantalum gauze was used in the repair of left inguinal hernia.

FIG. 8. X-ray flat plate of abdomen shows tantalum gauze well in place on the seventh postoperative day.

FIG. 9. Patient in standing position shows obliteration of hernial defect six weeks postoperativley.

tension on any of the supporting structures, that is using tantalum cloth or gauze as a patch to cover the hernial defect." Microscopically, it has been shown that the fibrous tissue encapsulates the gauze and becomes adherent to the surrounding fascia thus producing a new wall of fibrous tissue. Experimental observations after eight months showed that these results were obtained. So-called work hardening may allow the gauze to break but this does not appear to alter the strength of the hernial repair. (Figs. 1 to 7.)

Iason[8] concludes his chapter on incisional hernia, after presenting the various reparative technics, by saying, "In general, it may be stated that the prognosis is good in small hernias and poor in those in which constitutional diseases are prime factors."

A patient with incisional hernia was recently operated upon in whom the problem of widely separated edges was solved by the use of tantalum gauze.

CASE REPORT

L. A., a thirty-one year old male, was first admitted April 30, 1945, with a history and findings which would support a diagnostic impression of cholelithiasis. However, because of

surgery for a ruptured appendix elsewhere several weeks previously, a diagnosis of right anterior subphrenic abscess was considered more likely. The patient was operated upon May 4, 1945, using an upper right quadrant transverse incision, through which an anterior subphrenic abscess was encountered and drained. During the postoperative convalescence he developed a severe cough with large amounts of purulent, foul-smelling sputum. A fistulous drainage through the right lung was considered and supported by x-ray findings. Improvement after chemotherapy and postural drainage was sufficient to warrant discharge from the hospital on May 25, 1945. A chronic cough persisted with small amounts of sputum. Subsequent x-ray studies suggested the presence of a residual subphrenic abscess. For this reason further surgical exploration was advised. The patient re-entered the hospital April 15, 1946, and was operated upon April 23, 1946, through a transverse incision over the twelfth rib which was removed. A residual abscess beneath the right diaphragm was encountered and drained. He was discharged May 14, 1946, much improved. At this time evidence of an oncoming incisional hernia was perceived. The patient wore a corset binder until November 14, 1946, when he acceded to advice for surgical repair.

Operation for the relief of the hernia was performed under spinal anesthesia November 25, 1946. The scar was excised and the skin and subcutaneous tissues were dissected until

faseial margins were encountered. The hernial defect measured about 6 by 8 inches, the widest dimension being transverse. The upper fascial border was found at the costal margin. The lateral border was along the latissimus dorsi. The medial border was at the midline and the inferior border had receded to halfway between the umbilicus and pubis. Fat, omentum and remnants of peritoneum presented in the defect. Enough tantalum gauze to cover the defect was fitted in a double layer. No attempt was made to pull the faseial margins together which would have been impossible. After covering the defect with the folded gauze its edges were everted and pressed down. The rolled edges of the gauze were sutured to the fascia with interrupted linen sutures. A few interrupted sutures of the same material fixed the peritoneal remnants to the gauze. The subcutaneous fat was closed with interrupted sutures of No. 1 chromic and the skin was closed with interrupted black silk sutures.

The immediate postoperative condition was without incident. The patient remained in bed for seven days at which time roentgenograms of the operative site revealed the radio-opaque gauze to be well in place. (Fig. 8.) The skin sutures were removed and the patient was discharged on the twelfth postoperative day with no complaints. A corset binder is being worn until it is thought that healing is sufficiently well established to support the defect. Brief examinations without the binder show the hernia to be obliterated. (Fig. 9.)

SUMMARY

Herein is presented a preliminary report on the use of tantalum gauze in the repair of a massive incisional hernia with good immediate results as well as x-ray pictures and photographs of three additional patients treated similarly.

It is far too early to draw conclusions, yet it would appear that this form of reconstructive repair deserves further study especially in those patients who do not lend themselves readily to other methods.

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USE OF APONEUROTIC FLAP IN INGUINAL HERNIOPLASTY

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A NEW method of inguinal hernioplasty modifying Beach's[1] aponeurotic flap was introduced by the author's service at the Sydenham Hospital, New York, N. Y.

FIG. 1. Aponeurotic flap formation by two vertical incisions 2½ inches long united by a transverse incision at their upper ends.

Beach believed that by his method he "was able to replace the inguinal canal by an exit thus putting a gravitational obstacle in the way of recurrence." We have found that this method of gravitation was insufficient and that a more effective repair can be accomplished by suturing the true border of the conjoined muscles to Poupart's ligament.

An inguinal incision 3 inches long is made. The upper angle is retracted to expose the external oblique aponeurosis in a wide area. A vertical incision 2½ inches long is made through the external oblique aponeurosis from the mesial margin of the internal ring. Another incision is made from the lateral margin of the external ring approximately 2½ inches long with a slight oblique deviation to the lateral side in order to avoid encroachment on the anterior rectus sheath. The upper ends of these incisions are united by a transverse incision. (Fig. 1.) This aponeurotic flap is retracted downward exposing the ingui-

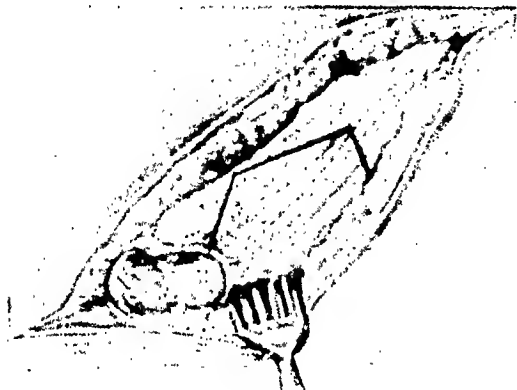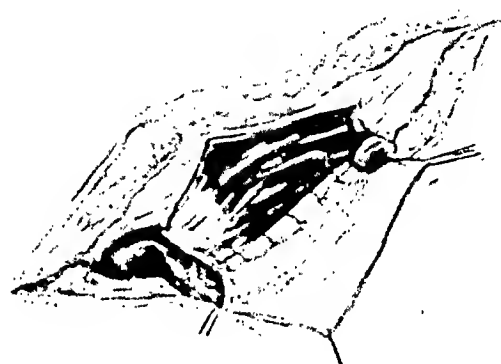FIG. 2. Application of Bassini suture upon excision of the hernial sac. The cord is transplanted extra-aponeurotically.

nal canal from above. The hernial sac is dissected from the cord, the highest point of its neck cleaned of surrounding tissue, transfixed and the sac removed. The aponeurotic flap is then passed beneath the cord. Poupart's ligament is swept clean of areolar tissue down to the tubercle of the pubic bone and the conjoined muscle border is sutured to its lower shelving edge by means of four or five interrupted chromic catgut sutures on an atraumatic
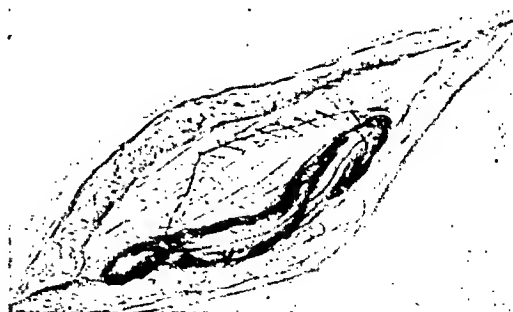

FIG. 3. Flap brought back to its former position and its cut edges approximated by a continuous catgut No. oo suture.

needle. The first suture is introduced through the periosteum of the spine of the os pubis and includes Gimbernat's ligament. (Fig. 2.) The flap is now brought back to its former position and its cut edges are approximated by a continuous chromic catgut No. 00 suture. (Fig. 3.) Two to three subcutaneous plain catgut sutures are introduced to obliterate the dead space. The skin edges are united by using Michel clips or fine silk.

COMMENTS

Since Lucas Champoniere, in 1883, first exposed the inguinal canal by splitting the external oblique fascia directly over it, many forms of repair have been described thus indicating that no perfect method has yet been attained. In each procedure scar tissue forms directly over the inguinal canal predisposing to atrophy and loss of elasticity of the fascia and affording the possibility of recurrences. We have attempted a more physiological approach to the repair of inguinal hernia by means of the flap method. The incision of the fascia is made about 2½ inches from the canal and therefore no loss of elasticity or atrophy can be expected. At the present time seventeen operations have been performed. These cases consisted of two sliding hernias, four direct hernias, two inguinal scrotal, one direct-indirect and eight oblique hernias. In each case we have found that the flap method afforded excellent exposure permitting us to treat adequately all varieties of inguinal hernias.

Although Beach's flap method of opera-tion may be adequate for young, strong individuals, we have utilized, in addition, the non-transplantation method of Coley-Ferguson; however, in cases of direct hernias, indirect hernias of long standing and wide-mouthed sliding hernias the flap method even with transplantation of the cord is insufficient to cure the hernia. In these cases the complete closure of the surgical triangle is obligatory. We have accomplished this by a Bassini suture and transplantation of the cord extra-aponeurotically.

Our results up to the present have been very encouraging. Postoperatively, the patients have a sense of well-being. Some have gotten out of bed on the second or third day and have been discharged on the seventh or eighth day.

SUMMARY*

1. A physiological approach to the repair of inguinal hernia has been described.

2. The method is simple and affords excellent exposure.

3. Hospitalization has been reduced from the usual ten to fourteen days to seven days.

The author wishes to thank Dr. Helmuth Nathan for his artistic drawings.

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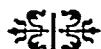
DIAGNOSIS OF ACUTE APPENDICITIS IN THE TROPICS

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THE onset of a number of diseases indigenous to the tropics is associated with abdominal signs and symptoms necessitating careful exclusion of acute appendicitis. For the surgeon whose judgment and experience as well as training are the product of a temperate climate the diagnosis of acute appendicitis under these circumstances presents a real challenge.

In a recent article Ravdin and Wolf[1] brought attention to the simultaneous occurrence of appendicitis and malaria as well as the difficulty encountered in interpreting abdominal signs and symptoms in patients with malaria and receiving antimalarial medication. Howe[2] has admirably discussed the problem in regard to amebiasis in his paper on the surgical aspect of intestinal amebiasis. During the past two years on the surgical service of a hospital operating in New Guinea and Luzon, P.I., we have encountered similar diagnostic difficulties in other tropical diseases as well.

While not exclusively confined to the tropics amebiasis is highly endemic in the Philippines. In the majority of cases encountered the possibility of appendicitis is not suggested although in cases with cramping abdominal pain, slight nausea and lower abdominal tenderness without diarrhea appendicitis must be excluded.

Recently a white male soldier was admitted complaining of nausea and lower right quadrant abdominal pain of twelve hours' duration. He had previously been in good health with no gastrointestinal complaints except for an attack two weeks previously of what he referred to as the "G.I.'s," an army term exclusively, which proved to be a bout of otherwise asymptomatic diarrhea which subsided completely in twenty-four hours. On examination significant findings were confined to marked lower right quadrant tenderness, rebound tenderness and muscle spasm. On rectal examination tenderness high on the right side of the pelvis was noted. A total leukocyte count of 16,500 with 82 per cent granulocytes and 18 per cent lymphocytes contributed to the provisional diagnosis of acute appendicitis. When the appendix was exposed and found to be normal in appearance the cecum was examined. The only lesion found was a granular appearing, hemorrhagic, acutely inflamed area 2½ cm. in diameter and sharply circumscribed on the lateral serosal surface of the cecum. The appendix was not removed and postoperatively active therapy for amebiasis was started. It was not until several weeks following operation that the diagnosis of amebiasis could be confirmed by the finding of endomoeba histolytica in the stool. Operation, in this instance, apparently did not alter the course of the disease.

Another case presented a similar syndrome of nausea, vomiting, and abdominal pain of sudden onset. Right lower abdominal tenderness, muscle spasm, tenderness on rectal examination and leukocytosis were found. The patient had had no history of diarrhea or other recent gastrointestinal symptoms. At operation a characteristic acute inflammatory process involving the appendix and the cecum for a distance of about 3 cm. from the base of the appendix was found. In this instance the appendix was removed because perforation appeared likely. Although active therapy for amebiasis was administered the patient subsequently developed a low grade hepatitis which prolonged convalescence for several weeks.

The explanation for the symptoms simulating appendicitis is not hard to find. In amebiasis the colon and occasionally the terminal ileum is invaded by the trophozoites of endomoeba histolytica. In some cases the cecum or, as noted above, even the appendix may be the site of the initial involvement. The primary lesion is a submucosal abscess which may go on to mucosal ulceration, thickening of the bowel wall and inflammation of the serosa.[3] In acute amebic typhilitis diarrhea may be

absent and the syndrome produced by such a localized lesion may be differentiated with difficulty from appendicitis due to other causes. Fortunately, in most cases diarrhea or more generalized findings are present. Trophozoites or amebic cysts may be found in the stool and when the rectum or sigmoid is involved characteristic proctoscopic findings are present.

"Dengue is an acute infectious disease caused by a filterable virus and transmitted to man by species of aedes mosquitoes."[4] Dengue and a large group of dengue-like fevers are endemic in the tropical islands of the Pacific. Generally, the diagnosis is easily made from the acute onset of the characteristic post-orbital pain, general malaise, joint pains and fever followed shortly by a morbilliform rash. Leukopenia is an outstanding finding and is helpful in making a diagnosis. Occasionally mild or atypical cases of dengue are encountered in which the onset and subsequent course of the disease lacks many of the diagnostic features. Mild abdominal soreness and tenderness associated with slight nausea frequently accompany the disease. Several patients with dengue in which abdominal soreness was the outstanding initial feature have been admitted to the surgical service. From these patients a history of onset several hours previously of slight general malaise, nausea, occasional vomiting and pain in the lower abdomen was obtained. In two instances tenderness and voluntary spasm in the right lower quadrant of the abdomen was of sufficient prominence to direct attention to the possibility of subacute appendicitis. In one instance right-sided tenderness on rectal examination was noted. In the absence of other signs the low initial leukocyte count with a relative lymphocytosis led us to defer operation. The onset several hours later of fever, generalized aching, severe headache and a few days later of a characteristic rash confirmed the diagnosis of dengue.

Although little is known of the pathology of uncomplicated dengue, a few petechial hemorrhages in the gastrointestinal tract and enlargement of the internal lymph nodes are mentioned by Stitt[5] as having been noted by other authors. Such findings could account for the signs noted in the cases mentioned.

Ancylostomiasis is widespread in the Philippines where it is usually due to infection by the helminth *necator americanus*. Considering the pathogenesis of the infection it can be expected that penetration of the mucosa of the small intestine by the worms will produce gastrointestinal disturbances. In patients encountered among the native population a recrudescence of hookworm infestation with abdominal pain and tenderness associated with nausea and vomiting has at times been confusing. The frequent necessity of obtaining a history through a lay interpreter coupled with a firm conviction on the part of the patient that he has appendicitis further complicates making a diagnosis. In spite of the fact that the duodenum and jejunum[6] are the site of the heaviest infection a number of instances have been encountered showing lower right-sided abdominal pain and tenderness with associated nausea. In such cases with a normal white count and relative eosinophilia operation has been deferred. The subsequent benign course of the disease and the finding of a hookworm ova in the stool have justified conservative therapy.

The large nematodes, including *ascaris lumbricoides*, *oxyuris vermicularis* and *trichuris trichuria*, are frequently found in the lumen of appendices removed in the tropics. In some cases acute appendicitis occurs as the direct result of their presence in the appendix. We have had two cases of acute suppurative appendicitis, one secondary to obstruction of lumen by a large ascaris worm, and the other due to the presence of a large number of oxyuris vermicularis. The mere presence of these worms in the bowel may give rise to ill defined abdominal symptoms which may be confused with appendicitis, but rather easily differentiated by examination of the stool for ova or worms. However, when the appendix

becomes obstructed or inflamed by these worms the resulting syndrome is not to be differentiated from appendicitis secondary to other causes.

In the tropics the constant presence of bacillary dysentery and multiple types of gastroenteritis is a cause of considerable concern to the conscientious medical officer. Occasionally the onset of diarrhea may direct attention away from the possibility of appendicitis only to have the persistence of symptoms and signs in the region of the appendix lead to operation. More often than not it will be found that the enteritis in the adjacent small bowel was responsible for the symptoms. However, a very acutely inflamed appendix was recently removed from a patient with an acute bacillary dystentery (Shigella paradysenteriae). Another patient was admitted with vomiting and diarrhea which had come on following a drinking bout. He ran a high fever, and passed fifteen to twenty blood-tinged stools daily. There was moderate leukocytosis. Treatment with sulfadiazine brought about little improvement in what was thought to be severe enterocolitis. Proctoscopic examination revealed only diffuse mucosal reddening. Stool examinations were not remarkable except for the presence of blood and pus. Ten days later a large left lower quadrant abscess was drained which subsequently was demonstrated to have been due to a ruptured appendix.

It is stated that the picture of subacute appendicitis occasionally develops in cases of schistosomiasis japonica.[7] In the early stages of the disease when the mature worms reach the intestinal tract and the females deposit eggs in the vessels of the intestinal wall, abdominal pain may be experienced. However, a history of exposure, the occurrence of fever, chills, urticaria, cough, generalized aching, leukocytosis with eosinophilia and the presence of ova in the stools should lead to the correct diagnosis. Schistosomiasis is not endemic on Luzon and we have had no direct experience with the disease in its acute phase.

SUMMARY

The presence of a number of tropical diseases new to the temperate climate surgeon stimulates interest in the differential diagnosis of appendicitis which too often is somewhat perfunctorily made. However, the principles of surgical judgment are no different in the tropics than any other place. The surgeon must familiarize himself with and also be prepared to recognize tropical disease which may simulate appendicitis.

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A TUNGSTEN STEEL GOUGE FOR USE IN A NICOLA OPERATION

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ANYONE who performs a Nicola procedure for recurrent dislocation of the shoulder realizes that technically
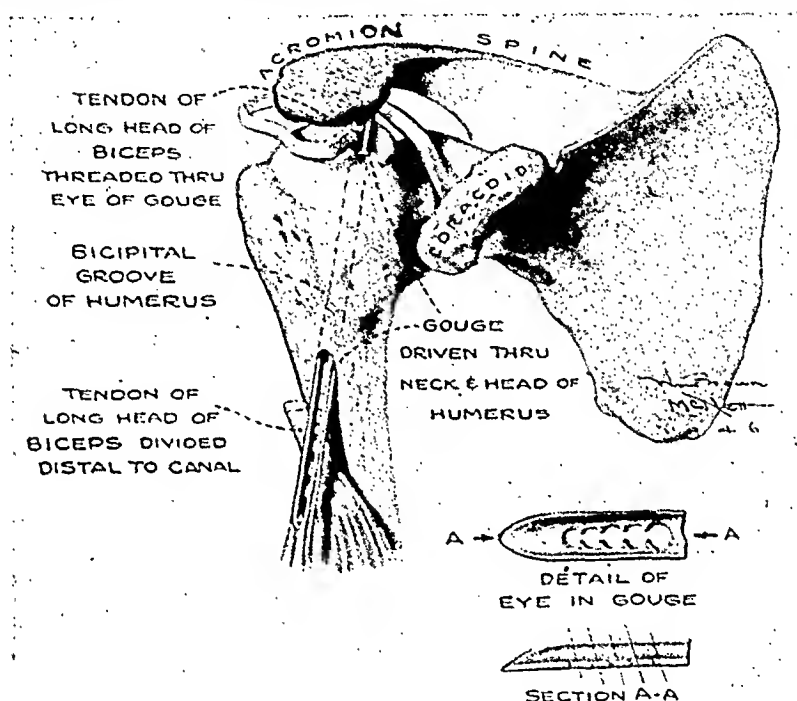

Fig. 1. This illustration demonstrates the use of the gouge.

a great difficulty is the proper placing of a drill hole so that it starts below the transverse ligament as it should and that it comes out at the center of the head. Another difficulty is in engaging the proximal end of the sectioned biceps tendon in a loop of wire, or whatever else may be used, in order to bring it down through this drill hole and suture it back to the distal portion of the long head of the biceps.

To simplify both of these details, it was believed that if a gouge along the lines of the one that Dr. Nicola used could be made, it would definitely aid in drilling the hole and controlling its position and point of emergence from the humeral head. For, if a long, sharp gouge is used it is easy to do the entire drilling and sight just the point on the head where the tip of the gouge should emerge. It was also believed that if in place of merely having an eye to thread a suture or wire through this gouge in order to pull the tendon down, holes might be so drilled that they would be oblique and in graduated size, the smallest at the tip and the largest toward the gouge handle. Then the tendon might merely be stuffed into the larger holes in the gouge and when the gouge is pulled down, the tendon would slide toward the smaller holes and be caught in the teeth-making of the edges of these continuous holes. The tendon would then come down with the gouge as it is withdrawn. It was realized that if the point of the gouge at this area was too thin it would shear the tendon, but it was believed that if it was thickened a little it would not injure the tendon in the least.

With this in mind a gouge such as has been described was constructed and to

obviate the possibility of breaking at the weakened point where so many drill holes were made it was felt that it would be best if it were made of a strong metal, such as tungsten steel. Such a gouge illustrated here was constructed and found to be very satisfactory. The technic is the introduction or gouge into the correct area in the region of the intertubercular groove and to drive it upward so that it will emerge at the center of the humeral head. Since the continuous connected holes are a little bit back of the point of the gouge, the point is merely continued upward into the acromion process and is partially buried in that structure. (Fig. 1.)

This then brings the slotted area of the gouge into clear view and accessibility, and it becomes a simple matter to take hold of the tendon end with a small hemostat and stuff its flat side into the long length of the drill holes until the tendon is stuffed through these holes. The gouge is merely withdrawn and since the larger holes are below the smaller holes, which are in turn at the tip or distal end of the instrument, the tendon merely rides up and engages upon the smallest holes and is tightly held so that it can be pulled through the entire drilled area with great ease.

The gouge measures ¼ inch in diameter and such a drill hole is adequate to bring the biceps tendon through the drill hole.

Correction: In our January, 1948 issue, on pages 234 and 235, in Figures 1 to 5, the author wishes to call attention to the fact that "After Holtfreter" should have been inserted under Figures 1 to 4 and Figure 5 to indicate the the original author of the illustrations.

VASCULAR OBSTRUCTED ACUTE GALLBLADDER

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IT is now almost generally agreed that the chronic condition of the gallbladder without stones had best be left alone surgically unless it is quite evidently a focus of infection or symptoms are severe and persistent. Conversely, all stones in the biliary tract are surgical problems even if they do not produce symptoms, since a high percentage of stones eventually cause serious complications.

That a cholecystectomy when possible is indicated rather than cholecystostomy, most all will agree. It is also agreed that when gallbladder surgery is being performed, if there is any possibility of their being a common duct stone present the common duct should be opened, probed, washed with saline and drained with a T tube. As yet no satisfactory way has been devised to deal with stones in the bile radicles in the liver itself; neither has an entirely satisfactory plan been devised for handling all patients with acute cholecystitis and lithiasis. Some have believed that all patients with cholecystitis and lithiasis should be operated upon as soon as possible. Others believe that the condition should be allowed to progress for several weeks and then an operation attempted. In some clinics and on some surgical services, rules have been promulgated to the effect that if an acute condition of the gallbladder is seen in the first thirty-six or forty-eight hours or sooner, the patient should be operated upon at once; if he is seen later, then he should be treated expectantly until a more appropriate time for surgery occurs. The reasoning behind this rule is that before forty-eight hours have elapsed the edema and inflammation is not sufficient to make handling of the ducts treacherous but that later than that it becomes so.

To the author none of the previous mentioned plans for acute conditions seem either satisfactory or based upon the pathological disturbance involved. In addition to the obstruction of the bile flow by stone, the important pathological condition which occurs and is frequently not even considered, is the vascular obstruction of the cystic vessels, either the vein or artery or both. This is, as a rule, due to the impaction of a medium size or large stone in the cystic duct. A condition results which is not dissimilar to that which occurs in an extremity when its main vessels are obstructed, namely, gangrene. Lesser degrees of vascular interference may cause a lesser degree of damage but with damaged tisssues infection readily sets in. Experimental infection of the gallbladder does not cause a pathological condition comparable to the above type of case.

That obstruction of the cystic duct or common duct alone causes no inflammatory or destructive action of the gallbladder wall, has been experimentally shown and is also clinically evident in patients with hydrops of the gallbladder as well as by the appearance of the gallbladder in those patients with carcinoma obstructing the common duct.

Infection quickly follows vascular involvement or sufficient local trauma in the gallbladder. The gallbladder of animals becomes infected by injecting bacteria into the wall of the gallbladder or by obstructing the common duct and injecting an organism into the gallbladder, although these do not proceed to rupture nor does gangrene develop. Of those clinical patients that have a ruptured gallbladder, gangrene, empyema, subdiaphragmatic or liver abscess as well as other severe and highly morbid states, most all had in the early stage a vascular obstruction due to the impaction of a stone.

In view of these observations it is obvious that to leave such a group of vascular obstructed gallbladder patients to "quiet down" is impossible. On the other hand to operate on patients with acute conditions who do not have vascular obstruction, after the forty-eight-hour period has passed, unnecessarily increases the risk and technical difficulty for these conditions will quiet down in time.

How then to determine which patients have a vascular obstruction? It is obvious that such patients must be operated upon as soon as hydration has been accomplished, even if an admittedly inferior operative procedure such as cholecystostomy has to be performed.

In the author's experience the persistence of pain is the predominating factor in differentiating these patients from those with simple colic or acute inflammation of a non-vascular obstructing nature. In addition, the early rise and continued height of the sedimentation rate is noteworthy. In the presence of persisting right upper quadrant pain, with a high sedimentation rate, the presence of a palpable, enlarged and tender gallbladder is further evidence of this condition.

If this conception of vascular obstruction of the gallbladder is kept in mind it will help us to recognize these pathological conditions early, so that the patients may then be operated upon early before other complications supervene.

SUMMARY

1. Patients with gallbladder disease plus vascular obstruction must be diagnosed early and operated upon as soon as possible.

2. Other patients with acute gallbladder pathological conditions should be allowed to quiet down before surgery, if seen forty-eight hours after the onset of symptoms; otherwise they should be operated upon at once.

3. The condition of the patients in the first group may be diagnosed by the persistent pain and increased sedimentation rate which is sometimes accompanied by a palpable, enlarged and tender gallbladder.

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Case Reports

TUMORS OF THE ADRENAL CORTEX

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THE adrenal gland has two component parts which are developmentally, histologically and functionally entirely unrelated.[1] Their importance in the scheme of life is shown by the fact that they have a more abundant blood supply than any other organ in the human body. It is interesting to note that the blood supply to the cortex and the medulla, which are totally unrelated in function, is the same.

Embryologically, the cortex is derived from the mesoderm and therefore is likey to form adenomas and adenocarcinomas. The medulla is derived from the same portion of the entoderm as the sympathetic ganglia and is therefore more liable to develop neuroblastomas, gangliomas and chromafinomas (phenochromocytomas).[2] In this paper aberrations of the cortex of the adrenal glands will be considered.

The secretions of the cortex are so numerous, the steroid hormones alone numbering twenty-five, that only a few of the more common secretions will be discussed. Desoxycorticosterone causes a marked retention of sodium chloride and water, increases the urinary excretion of potasium and phosphorous and causes elevation of the blood pressure. The hormones which affect carbohydrate metabolism are corticosterone, dehydroxycorticosterone and 17-hydroxy 11 dehydroxycorticosterone. The latter[3] is also known as compound E of Kendall or compound F of Pfiffner and Wintersteiner. It is also known that the adrenal cortex controls the excretion by the urine of the 17-ketosteroids (the androgen equivalent) and is responsible for the origination of the beta fraction.

Taken as a whole, the physiology of the adrenal cortex is concerned with: (1) electrolyte metabolism, (2) carbohydrate metabolism, (3) renal function, (4) growth of young animals, (5) resistance to stress and infection, (6) regulation of blood pressure, (7) muscular response, (8) sexual development, (9) vitamin C storage and metabolism and (10) lactation.

That there is an inter-relationship with all the glands of the body is clearly shown by the fact that diseases or tumors of the pituitary, ovary, thymus, pineal body and adrenal cortex may all give rise to identical clinical entities. These conditions are listed as follows:[3] (1) Tumors of the adrenal cortex. (2) hyperplasia of the adrenal cortex; (3) fuchsinophilic granules of the cells of the adrenal cortex; (4) basophil adenomas of the anterior lobe of the pituitary; (5) hyaline degeneration of the basophil cells of the anterior lobe of the pituitary; (6) sarcomatous and undifferentiated tumors of the anterior lobe of the pituitary; (7) arrhenoblastoma of the ovaries; (8) granulosa cell tumors of the ovaries; (10) Morgagni-Morel syndrome (osteitis frontalis interna); (11) tumors of the pineal body; (12) tumors of the thymus and (13) the classical picture is not infrequently observed in patients in whom no gross or microscopic abnormalities of any of the endocrine glands can be determined.

Any of the above conditions may give rise to three separate groups of symptoms

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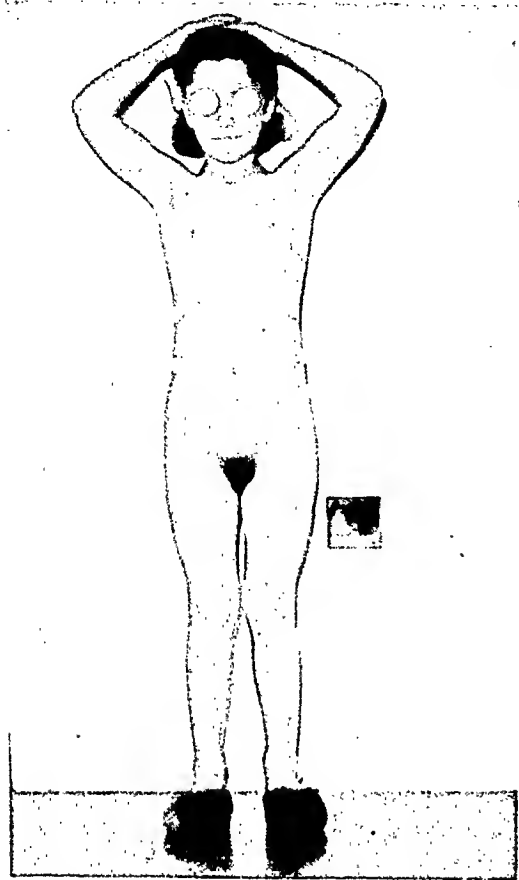
Fig. 1. General appearance of a girl of six years in Case 1.

Fig. 2. Typical female escutcheon found in Case 1.

which manifest themselves as follows: *First*, the production of premature sexual and physical development in children. *Second*, in adults an increased or decreased virility or alteration of sex characteristics. *Third*, the symptoms of obesity, hypertension, glycosuria, osteoporesis and cutaneous striations, which are called Cushing's syndrome. In the differential diagnosis of any one of these three symptom complexes, all but one of the various causative agents as listed above must be eliminated before establishing treatment.

In this paper three case histories will be presented which represent (1) hyperplasia, (2) adenoma and (3) adenocarcinoma. These three cases also illustrate each one of the three different clinical syndromes previously described.

The first case demonstrates how simple hyperplasia of the adrenal cortex may produce premature sexual and physical de-
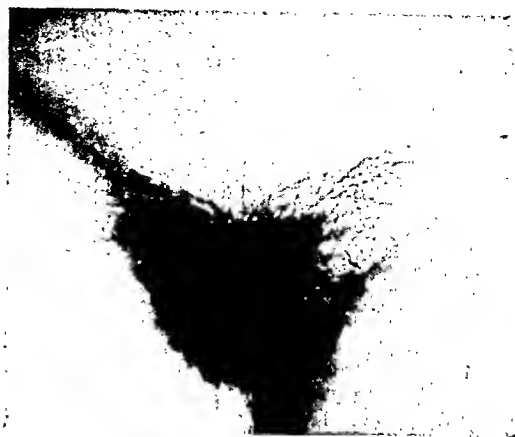
velopment in children with occasional alteration of secondary sex characteristics.

CASE REPORTS

CASE 1. S. S. (No. 2069, 2563, 3654, 3793.) The patient, a six year old, white dependent, was referred to the U. S. Naval Dispensary Pediatric and Gynecologic Departments by her school physician because of exhibitionistic tendencies. The physician discovered that this child, a muscular, unusually strong, boyish-girl, with a deep voice, who displayed impulsive, irregular sex behavior, was a possible pseudohermaphrodite. The clitoris was noted to be the size of a ten year old boy's penis and there was considerable pubic and some axillary hair. (Fig. 1.)

The girl was admitted to the U. S. Naval Hospital for study. She was found to be a sturdy, athletic child, active and alert under height for her age but not obese. There were no gross abnormalities of the skin, extremities or skeletal systems. The thyroid, heart and lungs were normal. There were no signs of breast development but axillary hair had appeared. The abdomen was also negative for masses, tenderness, etc., but there was well defined female escutcheon at the pubis. (Fig. 2.) The clitoris was noted to be 3.5 cm. long and 2.5 cm. at the base with a well formed glans resembling an hypospadiac phalus. Examination under ether anesthesia confirmed the presence of a normally deep vagina behind a snug hymeneal ring. The urethra was normal and a small cervix, uterus and normal ovaries were palpated. There were no masses in the pelvis.

Laboratory and other studies included the

following reports: I.Q. 99., basal metabolism minus 13 (satisfactory). Bleeding and clotting times 3½ minutes each; hemoglobin 91 per cent (Haden); red blood cells, 4.65 million; white blood cells 5,450, with 54 per cent polymorphonuclears, 41 per cent lymphocytes, 4 per cent monocytes and 1 per cent eosinophiles. These essential blood elements were always repeatedly similar to this report. The urine was invariably negative for sugar, albumin and the microscopic findings were not remarkable. The urine assay for the 17-ketosteroids was reported as 24 mg./24 hours (normal 4-6). X-rays of the wrists, etc., for bone age were reported as, "Characteristic of at least 10 years as compared with a chronological age of 6 years." Intravenous pyelograms, in the supine and erect positions, were, "Normal without evidence of pressure or mass at either upper kidney pole." Roentgenography of the skull and sella turcica was not remarkable. The pineal gland was not calcified or delineated.

The past history included a normal birth and no neonatal complications. Rubella, epidemic parotitis and tonsillitis were the only significant infectious diseases. There had been no operations or injuries. The child had always been bright and active.

The family history was significant only for hypothyroidism without goiter in the maternal grandmother and mother. Both had taken thyroid with benefit. There was one older sibling, a girl of ten years who was quite normal and feminine.

Diagnosis: Adrenogenitalism with pseudohermaphrodism (or pubertas precox.)

A bilateral exploration of the adrenals was affected under cyclopropane anesthesia with good exposure of both glands. The right was average in size and appearance. A liberal biopsy was obtained. The left was larger than the right and displayed a nodular, lobulated appearance with what the pathologist termed a mild degree of hyperplasia. No tumor was felt or seen on section of the glands. Approximately one-third of this adrenal was removed for pathologic study and whatever therapeutic benefit might accrue.

The patient was supported while under surgery although she slipped into mild shock. She responded well to therapy without eschatin which was kept in readiness. A moderately severe adynamic ileus postoperatively was combatted successfully with oxygen, para-

Fig. 3. Greatly hypertrophied clitoris in Case 1.

sympathetic stimulants, Wangensteen suction and enemas. The patient recovered despite rather extreme respiratory distress which was abdominal and bronchitic in origin, roentgenography of the chest revealing no pneumothorax, fluid, pneumonia, etc. There were three days of low-grade fever. The incisions healed *per primum* and she was discharged as recovered on the eleventh postoperative day.

The child was readmitted to the Hospital after four months' observation during which time no change in the voice, habitus, clitoris or behavior were noted. An excision of the clitoris was accomplished under cyclopropane anesthesia. She was discharged after nine days' hospitalization; the incision healed by first intention and with a good cosmetic result.

The pathological report revealed: "Normal appearing adrenal tissue. The zona glomerulosa cells contain a normal amount of lipoid material. The zona fasciculata and zona reticularis show no pathology. The left adrenal presents a similar picture to the right with no evidence of pathology. Impression: 'Normal Adrenals.'

"The clitoris measuring 3.5 x 2.5 cm. (glans 1.4 x 1.2 cm.) is obviously enlarged with hypertrophy of the smooth muscle elements within the muscular wall." (Fig. 3.)

The follow-up one year after adrenal explora-

FIGS. 4 and 5. Case II prior to surgery demonstrating the distribution of fat and hair.

tion revealed slight growth of the child in stature. She was still agressive, athletic and muscular, with a boyish trend. An even more profuse pubertal hair pattern had developed. The site of the clitoris was normal. An essentially male psyche persisted but there was definitely less overt behavior, aberration and exhibitionism.

The second case of adenoma in a young woman demonstrates the second group of symptoms in which there is alteration of the secondary sex characteristics in adults.

CASE II. I. L., age twenty-six, was admitted to the hospital in February, 1945, complaining of amenorrhea of three years' duration. The onset of menses was twelve years; she had a regular twenty-eight-day cycle, of two to three days' duration with dysmenorrhea. She had an appendectomy in 1940, at which time a small ovarian cyst was ruptured but not removed. Following this the interval between menstrual periods became progressively longer and finally in November, 1942, menses ceased entirely. There had been no vasomotor changes, no headaches and no change in weight but there was slight blurring of vision which was not constant. There was no nocturia, polyuria or change in appetite. About June, 1943, she noticed that hair was growing on her face, breasts, abdomen and legs. This became progressively worse until she had to shave her face once or twice daily and chest and legs weekly. There was also a gradual deepening of the pitch of her voice.

She had been adopted in infancy and knew nothing of her family, although there were several brothers and sisters. She had uncomplicated mumps, chicken pox and pertussis in childhood. Tonsillectomy was performed in 1928 and appendectomy in 1940.

This young woman, 5 feet 1 inch tall, weighed 120 pounds; temperature 98°F., pulse, 20, blood pressure 120/90. (Figs. 4 and 5.) The pupils were round and regular; fundi and discs were normal; the tonsils were surgically absent; the thyroid gland was not enlarged. There was coarse hair on the face, chest, breasts, thighs and abdomen of a masculine distribution. There was evidence of recent shaving of the upper chest and face.

The chest was symmetrical with breasts fairly full and well rounded. The heart and lungs were normal. The abdomen revealed no masses or tenderness. There was a long right rectus incision from a former appendectomy.

The genitalia were normal except for an enlarged clitoris. The hymen would not admit the examining finger. The consultant in gynecology made an examination under anesthesia which was negative for tumor and which showed the presence of normal tubes, ovaries and uterus. Currettings obtained at the same time showed only mucoid material. The neurologic examination was negative. Laboratory findings: Kahn—negative; several specimens of urine negative, red blood count 4,250,000, hemoglobin 80 per cent white blood count 5,600 segments 55, lymphocytes 32, bands 3. A glucose tolerance test using 50 Gm. of glucose revealed a flat curve reaching a high of 111 mg. per 100 cc. and low of 83 mg. with no sugar appearing in the urine. Further laboratory examination revealed blood calcium, 9.3 mg. per cent, blood phosphorous, 5.4 mg. per cent, blood chlorides 627 mg. per cent. The basal metabolism was plus 3; an electrocardiogram was normal. A forty-eight-hour specimen of urine was examined and showed 93.8 mg. of total 17 ketosterones (androsterone equivalent) per 24 specimen. (Normal in the adult female is 8 to 13 mg per twenty-four-hour specimen); estrogen twenty-four-hour, 25 units. (Normal 16 to 20.) A salt tolerance test made by giving 10 gm. sodium chloride in 1,000 cc. of water and testing the urine hourly showed normal excretion.

X-rays of the sella turica and chest were negative. Intravenous pyelogram and steroscopic studies of the kidney and lumbar area showed a negative urinary tract with a somewhat oval shadow about 6 by 5 cm. immediately above and overlapping the pole of the right kidney, suggestive of a tumor mass in the right adrenal area. A perirenal insufflation test was not done.

She was operated upon May 14, 1945, under continuous spinal anesthesia plus sodium pentothal intravenously as an adjunct. The left side was entered first to determine the presence of a normal adrenal there. This is essential as death would ensue if the adrenal were to be removed from one side with a congenital absence or inadequate gland on the other side. When the left adrenal was found to be present and normal, the right side was opened. The twelfth rib was resected and two tumors of the adrenal exposed. One was oval, 5 cm. in diameter, and the other 2½ cm. in diameter. They were loosely adherent to the diaphragm. The three vessels supplying them were ligated and the tumors removed. The wound was closed in layers with a small rubber tissue drain. She received 500 cc. of citrated blood during the operation and 1,000 cc. of 5 per cent glucose in saline.

The pathologic examination of the gross specimen showed two adjacent, encapsulated, soft, reddish-brown masses, one about 5 cm. in diameter and the other 2.5 by 2 by 2 cm. There were many irregularly shaped yellow foci in the capsules. The cut surfaces were soft, reddish and yellowish, and moist. There were a few hemorrhagic foci.

Microscopically, the tissue was composed of anastomosing cords and solid sheets of polyhedral cells resembling those of the zona fasciculata and reticularis of the adrenal cortex cytoplasm; others were granular. The nuclei tended to be central and round; and while there was some variation in size, the tendency was to uniformity. No mitoses were seen. There were scattered foci of hemorrhage but there was little pigmentation and no necrosis. Intravascular growths were not seen and there was little stroma.

Diagnosis: Adrenal adenoma; no histologic evidence of malignancy.

Postoperatively she received 1 cc. of eschatin every four hours. The blood pressure was taken every half hour with orders to give an extra cc. of eschatin each time it dropped below 80 systolic. This happened only once, on the first postoperative day, and during the first three days the systolic pressure ranged between 96 and 103 without rapid drops or rises. On the third day she had some distention which was controlled by enema and prostigmin. The highest rise in temperature was to 101°F., pulse 112, respiration 24 which rapidly subsided. Two days postoperatively the blood calcium was 9.7, phosphous 5.1, sugar 65.2 and the chlorides 627 mg.

On the seventh day the sutures were removed and the wounds healed except for a small draining area in the right side which subsequently healed well. Seven days postoperatively the blood calcium was 8.8 mg. blood chlorides 479.5 mg., phosphorous 3.9 mg. and the sugar 93.8 mg.

The urine was normal; red blood count 4,400,000; hemoglobin 77 per cent; white blood count 9,000; segments 66 and lymphocytes 34. On the twelfth postoperative day she

6   7

FIGS. 6 and 7. Case II fifteen months after surgery and three months after childbirth showing change in distribution of hair and fat.

developed a mild, superficial thrombophlebitis of the left leg which cleared up under penicillin treatment.

On July 12, 1945, a re-examination of the urinary output of hormones showed androgen 8.6 units compared with the 93.8 mg. preoperatively. (Normal 15 to 20 units.) The estrogen was also low showing 5 units with 12 to 16 units as normal. The hair on the face and breasts disappeared; pitch of voice became higher and, although there was no weight loss, the fat became redistributed so that the general appearance was different and she appeared quite feminine. The clitoris remained unchanged.

She returned to the East and married her childhood sweetheart from whom she had been estranged during her period of masculinity. A recent communication announced that she now is the mother of a normal baby girl. (Figs. 6 and 7.)

The third case, one of adenocarcinoma of the adrenal cortex, demonstrates that a very large tumor of the adrenal may give rise to a very few symptoms. The symptoms that are present fall into the classification of Cushing's syndrome.

CASE III. Mr. G. F. age fifty-one, was admitted to the hospital July, 1946, complaining of vague indigestion and epigastric pain unrelated to meals. The pain was severe and accompanied by dizziness, sweating and rise in temperature to 102°F. He had tarry stools for about four weeks and, although he weighed 180 with a height of 5 feet 6 inches, claimed to have lost 20 pounds in two months. Sexual habits were unchanged.

Examination revealed a sthenic type of middle aged, obese male; temperature 99°F., pulse 80, respirations 20, blood pressure 110/75. All systems were normal except for tenderness over the epigastrium; an enlarged liver could be palpated four fingers below the costal margin. There was a questionable mass in the right side which could not be distinguished from the enlarged liver. X-ray examination of the gallbladder was negative. The gastrointestinal series showed a large diverticulum of the duodenum with some associated irritation in the descending duodenum. On flouroscopic exami-

Fig. 8. Gross specimen of tumor in Case III; weight 1,539 Gm.

nation there was tenderness to pressure directly over this area. Laboratory examinations revealed red blood cells 4,000,000; hemoglobin 12 Gm. white blood cells 15,000; segments 64, lymphocytes 30, bands 5, eosinophiles 1; repeated urine examinations were negative; non-protein was 20 mg. per cent.

He was operated upon on August 18, 1946, through a right rectus incision for correction of the duodenal diverticulum. He was given spinal anesthesia consisting of 100 mg. novacain and 10 mg. of pontocaine. The diverticulum was closèd by a series of purse-string sutures and the abdomen explored. The liver was smooth and uniformly enlarged. A soft mass was found between the right kidney, liver and the diaphragm measuring about 25 by 15 by 10 cm. The left adrenal was located and inspected to make sure it was present and the mass on the right removed. The blood vessels were very engorged and the branches from the phrenic vessels difficult to control. A fibrin foam pack was put against the diaphragm and held there by gauze packing and the abdomen closed. The gauze was removed gradually from the third to the fifth postoperative days.

During surgery he received 500 cc. of citrated blood and 1,000 cc. of 5 per cent glucose in saline. For the first three days he was given 1 cc. of eschatin every four hours as prophylaxis against shock from the removal of the adrenal. It is estimated that it takes about three days for the remaining adrenal to take over the function of both. His postoperative course was smooth except for some coughing. His blood

pressures remained between 108/68 and 126/88. An x-ray survey of all his bones, which was made in search for metastases, was negative, as was also the chest x-ray.

The pathological report was as follows: gross: The specimen consisted of encapsulated ovaloid tumor mass measuring 18 by 14 by 14 cm. The tumor was soft in consistency and slightly lobulated. Cut section of one-half of the tumor showed the central area to contain a spongy-like, yellowish necrosis in center of which were several large blood vessels. Around the periphery was a layer 1 inch thick of somewhat softer, friable tissue of pinkish color. The tumor weighed 1,539 Gm. (Fig. 8.)

Microscopically, the sections revealed a rather varied picture. In some areas there was almost exact duplication of adrenal cortex. In these areas there were cords of alveoli masses of cells with abundant foamy, pink cytoplasm and vesicular round to oval nuclei. These masses were separated in general by a thin band of fibrous tissue. Some areas showed pseudopapillary arrangements with a central core of fibrous tissue resembling hypernephroma. In most of these areas the cells were regular and showed no marked anaplastic activity. In other portions, however, the alveolar and cord arrangement of cells was lost and there were broad sheets of diffuse cellular proliferation with little or no fibrous tissue stroma. Where the individual cells showed marked anaplasia and undifferentation, most of the cells were of large irregular hyperchromatic nuclei some of which were solid and some vesicular and there were

large sections of necrosis. The portion of the tumor of yellowish color seen in the gross represented necrotic tissue. This tumor in all probability represented malignant degeneration of a previously cysting adrenal cortaladenoma.

Diagnosis: Adenocarcinoma, adrenal.

The patient returned to his regular work one month after surgery and has been completely symptom-free for thirteen months.

COMMENTS

Case I. It would seem that Case I represents an instance of pubertas praecox, due to hyperplasia of the adrenal cortex, and is but one step beyond the "constitutional type" of sex precosity of the adrenal type. (In the constitutional type no abnormality, either gross or microscopic, is disclosed and the condition is considered due to hyperfunction. Unfortunately, resection of the adrenal gland alone does not appear to be a solution to the problem, nor is irradiation suited to these cases. The closest we can come at this time to remedial therapy in the pseudohermaphrodite group, following surgical diagnostic measures, is to remove the hypertrophic clitoris which would appear to be the focus for sex preoccupation and the site of interest in the exhibitionistic behavior.

The correct supervision of sexually precocious children is most important to prevent advances and indulgence by more mature members of the opposite sex and to avoid venereal disease. The risk of psychic trauma and even pregnancy in the more adult girls is obvious. The cooperation of the patient, patient's parents and physician is vital in this social-medical problem.

Case II. This case illustrates how all of the secondary sex characteristics can be altered by a relatively small tumor of the adrenal cortex. What is more important, however, is that these changes are all reversible (with the possible exception of regression in size of the clitoris) in a short time following surgical removal of the tumors and the patient can be restored to society as a normal individual capable of motherhood.

Case III. In contrast to Case II this tumor was enormous, but showed so few symptoms that it was a secondary condition which led the patient to seek medical care and to the discovery of the cortical tumor which was found to be malignant. Surgical extirpation was the only type of treatment to be considered.

SUMMARY

Three cases of adrenal cortical disease have been presented. These three demonstrate every variety of symptom complex which has sometimes been called the adreno-syndrome with the last case falling into the category of Cushing's syndrome.

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PRIMARY CARCINOMA OF BARTHOLIN'S GLAND*

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OF the malignancies of the female genital tract, carcinoma of the Bartholin or vulvovaginal gland is the one most seldom recorded. In this article I wish to add a case of ours and bring the number of recorded cases up-to-date.

CASE REPORT

Mrs. C. J., a widow aged sixty-four, was first seen by us on February 15, 1929. Items of importance in the past history were that she had six full term pregnancies and had two operations, a kidney stone was removed in 1923 and her gallbladder was drained in 1924. Her chief complaint on this first visit was that her periods, which had always been regular, had for the past nine months become irregular and profuse. A curettage was advised and this was done on February 23, 1929. The pathologic report disclosed endometrial hyperplasia. Following this curettage, her periods remained normal until March, 1930, at which time they stopped entirely.

The patient failed to return for check-up examination, as advised, and was not seen again until January, 1941, at which time she had a recurrence of vaginal bleeding. Another curettage was performed and adenocarcinoma of the endometrium was found. She was immediately given 3,000 mg. hours of intra-uterine radiation and deep x-ray therapy; two weeks after completing x-ray therapy, a complete hysterectomy and bilateral salpingo-oophorectomy was done. Regular examinations since have revealed no evidence of recurrence of the endometrial carcinoma.

On April 25, 1944, the patient appeared with the complaint of vulvar itching of one months' duration. On examination a solid growth of the right vulvovaginal gland was found. The growth was roughly spherical and was about ½ inch in diameter. On the surface was a small ulcerated area. The whole mass was partially movable and, although it extended deeply, it was not fixed to the ramus of the pubic bone. There was no evidence of recurrence of the uterine carcinoma in the deep pelvic structures in the vagina. There was no evidence of involvement of the inguinal glands. The pre-operative diagnosis was carcinoma of Bartholin's gland. Her previous pelvic check-up examination on November 22, 1943, had shown no evidence of trouble in the vulvar area. Figure 1 shows the gross and Figure 2 shows the microscopic evidence of the pathologic condition.

On May 8, 1944, a complete vulvectomy was done, care being taken to excise well beyond the infiltrated area of the growth. The patient was not a good operative risk and this, plus the fact that she evidently had a tendency toward carcinoma, we believed contraindicated the usual radical gland resection.

The patient was seen last on November 18, 1947, and there was no evidence of recurrence.

COMMENTS

The earliest reported case of carcinoma of Bartholin's gland was that of Kolb in 1864. Sixteen years later in 1880 Sinn recorded the second case. In the next thirteen years cases were recorded by Geist, Schweitzer and Mackenrodt. In 1906 Gaston Chaboux wrote an excellent thesis on carcinoma of Bartholin's gland and tabulated ten cases reported up to that time, including one of his own. Falls reviewed the literature up to 1923 and recorded a total of seventeen cases in table form. In 1930 Schneider brought the record up-to-date and found thirty-seven cases in all of proven primary Bartholin gland carcinoma. The next summary in table form was published by Simedinger in 1939; he collected thirty-eight cases. Some of those previously recorded by Schneider do not appear in his report. The most recent report in tabular form was made in 1944 by Aquinaga of Brazil. The recent reports of Boynton and Cosbie and some of the cases

FIG. 1. Specimen from the vulvectomy. In region of the right Bartholin gland there is a raised, firm area 1 cm. in diameter with superficial ulceration as shown. The clitoris can be seen above in midline.

FIG. 2. The section shows a definite adenocarcinoma of the Bartholin gland. There are many anaplastic cells but there is as yet no evidence of invasion of the basement membrane and very few mitosis. A moderate inflammatory cell infiltration is present.

in Simdinger's table are not recorded in Aquinaga's table. (Table 1.)

Undoubtedly there are some proven cases not recorded in the literature and also probably some in which the diagnosis was missed. The absence or confusion of pathologic details in the early reports and, in a few of the later ones, makes critical comment as to the validity of the diagnosis impossible.

A review of statistics from numerous large clinics show that carcinoma of the vulva comprises 3 to 5 per cent of all carcinomas of the female genital tract and

carcinoma of Bartholin's gland 2 to 3 per cent of vulvar carcinomas. In our 18,745 private obstetric and gynecologic patients there were thirty-one patients with vulvar carcinoma, one of whom had carcinoma of Bartholin's gland.

chronic infection or removal of a small cyst.

Honan gave four diagnostic criteria for carcinoma of Bartholin's gland: typical vulvar site, position deep in the labia, connection with the gland duct and presence of intact gland tissue. Shaeffer states

TABLE I
CASES REPORTED SINCE AQUINAGA'S TABLE

Author	Age	Previous Infection	Operation Radiation	Microscopic Pathology	Inguinal Metastasis	Result
1. Boynton.....	54	Swelling 6 mo.	Excision wide; x-ray, second excision after 5 yr.	Squamous	None	Recurrence in 5 yr.; died 1 yr. later
2. Sperl.........		Reported case in *Wien. med. Wchnschr.*, 1943		Not available for details		
3. Cosbie.......	59	Soreness 3 mo.	Refused operation; given x-ray; for recurrence in 3 yr. 200 mg. hr. radium; 1 yr. later radon seeds	Basal cell carcinoma	3 yr. later; metastases, lungs, spine, long bones later	Died 7 yr. after first visit
4. Crossen, R. J.	64	Itch 1 mo., radium and panhysterectomy for an adenocarcinoma fundus 4 yr. before; no recurrence	Vulvectomy wide	Adenocarcinoma Bartholin gland primary	None	No recurrence 3½ yr.

TABLE II

	3 Yr.	4 Yr.	5 Yr.	6 Yr.	7 Yr.	16 Yr.	At Last Report	
Excision wide........................	1L	1L 2D	1L	1L	1L	1L	6L	2D
Excision radiation.....................	1L	1L	..	1D	..	..	2L	1D
Radiation x-ray.......................	..	..	..	..	1D	..	...	1D
Vulvectomy...........................	1L	..	..	..	..	..	1L	
Vulvectomy gland resection.............	..	1D	..	..	..	..	...	1D
Vulvectomy radiation x-ray.............	..	..	1L	..	..	..	1L	
Vulvectomy gland resection radiation......	1L	..	..	..	..	..	1L	
							11L	5D

L—living at time of report.
D—dead at time of report.

The diagnosis of carcinoma is frequently missed preoperatively and the true nature of the lesion becomes evident only after an incision has been made for drainage of

that if the skin is intact and the growth is a frank adenocarcinoma, it is proof of the primary site. When the carcinoma arises from the duct, the differential diagnosis

from squamous carcinomas of the vulva is difficult as there is usually some metaplasia of the duct epithelium. In order to clinch the diagnosis, according to Frank and Kehrer, one must be able to trace the continuation of the carcinoma into the duct epithelium. Babson and Meeker believe that the finding of transitional epithelium of the communicating ducts is significant as this occurs on the vulva only in Bartholin's gland. Hidradenoma of the vulva is sometimes mistakenly diagnosed as carcinoma of the vulvovaginal gland.

The case of the youngest patient, nineteen years old, was reported by Beekman and the oldest ninety-one years old, was reported by Pape. Eichenberg in reporting 174 patients with carcinoma of the vulva found the average age to be 58.8 years.

Of the eighty-eight patients reported in the literature there were seventy-three whose age was given. Bartholin carcinoma was found to occur most commonly between the ages of forty to sixty. Twenty-two per cent occurred under the age of thirty and almost 25 per cent occurred after sixty. Hence, carcinoma must be considered in the differential diagnosis in any patient with enlargement of Bartholin's gland.

The signs and symptoms in order of frequency were: a tumor, usually cystic and in some cases painful; an abscess or draining sinus and swelling with soreness or itching.

The enlargement varied from a pea-sized tumor to one the size of an orange; the latter had been present for two years. There were two other patients in whom the tumor had been present for a year, one the size of a lemon and the other as large as a cherry. Rabson and Meeker found that patients had symptoms for an average

of fifteen months before consulting a physician.

Treatment varied from wide excision, vulvectomy, vulvectomy with gland resection, radiation therapy either with radium or x-ray and combinations of these treatments.

Of the eighty-eight patients, thirty-seven had no report of their final outcome and there were four cases of sarcoma. Of the remaining forty-seven, nineteen were followed to death or recurrence and, of these, fourteen either died or had recurrences within three years of treatment leaving five of this group who survived longer than three years.

In the group of patients alive and with no recurrence at the time of the report, seventeen had survived less than three years and eleven were alive without recurrence more than three years after operation.

Of the eight patients surviving past three years, five had adenocarcinoma, two had squamous cell carcinoma and in one the type was not determined.

In regard to prognosis, no conclusion can be drawn because of the small number of patients involved.

The type of treatment used in the sixteen patients surviving more than three years after operation and their survival times are shown in Table II.

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CARCINOMA IN EXSTROPHY OF THE BLADDER*

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THIRTY cases of carcinoma occurring in the exstrophic bladder have been reported in the literature. We wish to report an additional case, making a total of thirty-one reported cases.

McCown[16] summarized the reported cases of exstrophy of the bladder in 1940 and added one case, bringing the number of reported cases up to twenty-five. He did not include the case reported by Montpellier in 1935.[17] In 1943, Abeshouse[1] reviewed the entire subject and added one case. Graham[6] reported two cases in 1942 and Etherington-Wilson[5] reported the thirtieth case in 1945. The addition of our case makes a total of thirty-one reported cases of carcinoma occurring in the exstrophic bladder. (Table I.)

CASE REPORT

The patient, (No. 25086B) a sixty-three year old, white farmer, was admitted to the hospital for the first time on August 6, 1946, with the complaint of hemorrhaging from a mass on the surface of an exstrophy of the bladder. The tumor had been present for over a year. The past history was essentially negative except for the existence of the exstrophy of the bladder since birth. The family history was markedly positive for diabetes mellitus as it was reported that all of his seven brothers and eight sisters had had diabetes mellitus.

On physical examination his temperature by mouth was 98.6°F., weight 128 pounds (usual weight 150 pounds); height 5 feet 6 inches; pulse 78 per minutes and totally irregular; blood pressure 120/76; respirations 18 per minute and regular. The patient did not appear acutely ill. The physical examination was negative except for the following features: There were a few inspiratory rales at the bases of the lungs posteriorly. The heart was fibrillating and there was a loud blowing systolic murmur present over the apex; the umbilicus was absent;

the bladder was exstrophic and the left two-thirds of its surface was covered by a friable, bleeding mass which obscured the left ureteral orifice. The right ureteral orifice could be readily seen and urine was noted coming from it. The penis and scrotum were small, the urethra epispadiac. There was wide separation of the pelvic bones with absence of the symphysis pubis. There were varicose veins of both lower extremities.

Laboratory findings were as follows: Urine from the right ureter showed no albumin, no sugar. The left ureteral orifice was obscured by the neoplasm. The non-protein nitrogen was 44 mg. 100 per cc. of blood; hemoglobin 64 per cent; red blood cells 4,300,000; white blood cells 8,100; with 83 per cent polymorphonuclear leukocytes. The Kahn test was negative. X-ray of the chest revealed no pathological condition. An intravenous pyelogram was done which showed normal filling of the calyces and pelves of the kidneys bilaterally but the left ureter was shown to be dilated. X-ray of the pelvis showed wide separation of the pubic bones and absence of the symphysis pubis. Biopsy of the tumor of the bladder was reported by the pathologist as being adenocarcinoma.

The patient was prepared for operation. An attempt was made to clear the colon by administering succynlsulfathiazole by mouth, cleansing enemas and low residue diet. On August 21, 1946, the ureters were transplanted into the sigmoid section of the colon by the Coffey 1 technic and the bladder was excised.

The postoperative condition of the patient was good. Chemotherapy and intravenous fluids were continued.

On August 23rd, the non-protein nitrogen was reported as being 84 mg. per 100 cc. of blood. He appeared mentally confused. The abdomen was distended and was treated by stomach tube and constant suction. On August 24th, his condition appeared very poor. A right pyelotomy was done at which time the right kidney pelvis and the right ureter were distended with urine. A mushroom catheter was

TABLE I

CASES SUMMARIZED BY McCOWEN[16] AND THE SUBSEQUENTLY REPORTED CASES

Case	Year	Author	Sex	Age	Lesion	Treatment	Comment
1	1895	Bergenhern (quoted by Lacene and Hovelaque)	M	35	Adenocarcinoma	Transplantation of ureters; extirpation	Working 6 months after operation
2	1901	Ehrich (*Beitr. z. klin. Chir.*, 30: 581, 1901)	F	44	Adenocarcinoma	Resection transplantation of ureters and bladder mucosa	Died 8th day from ascending infection
3	1904	Enderlen (*Verbandl. d. deutsch. path. Gesellsch.*, 7: 167, 1904)	F	38	Adenocarcinoma	Radio therapy	Not benefited
4	1905	Lampe (*Verbandl. d. deutsch. Gesellsch. f. Chir.*, 34: 226, 1905)	M	..	Squamous cell carcinoma	Sonnenberg operation	Recovered; not treated
5	1906	Von Elselberg (*Wien. klin. Wchnschr.*, p. 548, 1906)	F	54	Adenocarcinoma	Extirpation	Living 15 months after operation
6	1909	Sargent (*Brit. J. Child. Dis.*, 6: 115, 1909)	M	39	Clinically, carcinoma	None	Not traced
7	1910	Wagner (*Deutsche Ztschr. f. Chir.*, 101: 330, 1910)	M	49	Adenocarcinoma	Biopsy only	Not traced
8	1910	Hager (*München. med. Wchnschr.*, 57: 2300, 1910)	M	66	Adenocarcinoma	Symptomatic	Died from cachexia
9	1910	Schloffer (*Wien. klin. Wchnschr.*, 23: 1158, 1910)	M	46	Carcinoma	Curettage	Result not mentioned
10	1912	Lacene and Hovelacque (*J. d'urol.*, 1: 493, 1912)	M	48	Adenocarcinoma	Symptomatic	Died from cachexia
11	1912	Lacene and Hovelacque, ibid.	F	26	Adenocarcinoma	Not mentioned	Result not mentioned
12	1914	Hunner (In Kelly and Burnam: Diseases of the Kidneys and Bladder. New York, 1914. D. Appleton & Co.)	F	26	Adenocarcinoma	Not mentioned	Result not mentioned
13	1918	Geraghty (In Cab: Modern Urology, Philadelphia, 1918. Lea & Febiger)	..	48	Adenocarcinoma	Not mentioned	Result not mentioned
14	1921	Lower (*Am. Surg.*, 73: 354, 1921)	M	50	Clinically, carcinoma	Transplantation of ureters; excision	Died; abdominal metastases
15	1922	Scholl (*Am. Surg.*, 73: 365, 1922)	F	23	Adenocarcinoma	Transplantation of ureters; excision	Died 2 years after operation from metastases
16	1922	Scholl, ibid.	M	48	Adenocarcinoma	Excision; radium	Reported well 6½ years later
17	1922	Dupont (*J. d'urol.*, 13: 433, 1922)	M	38	Adenocarcinoma	Exclusion of rectum	Died 2 days after operation
18	1924	Murphy (*J. A. M. A.*, 82: 784, 1924)	M	49	Adenocarcinoma	Radium emanation	Examined after 14 months
19	1925	McCarthy and Klemperer (*J. Urol.*, 14: 419, 1925)	M	47	Adenocarcinoma	Diathermy; ureterostomy; nephrectomy; cystectomy	Convalescent at time
20	1928	Judd and Thompson (*Arch. Surg.*, 17: 644, 1928)	F	38	Adenocarcinoma	Cautery excision of bladder	Well 3 years after operation

TABLE I (*Continued*)

Case	Year	Author	Sex	Age	Lesion	Treatment	Comment
21	1929	Hammer (*J. d'urol.*, 28: 260, 1929)	M	60	Adenocarcinoma found on Maydl block 10 years after above operation	No treatment; uremic death	No metastases found
22	1929	Hammer (*J. d'urol.*, 28: 260, 1929)	F	55	Adenocarcinoma	Uremic death	No metastases found
23	1930	Scheuer (*Ztschr. f. urol. Chir.*, 30: 299–308, 1930)	M	39	Adenocarcinoma	Removal electrocautery; x-ray therapy; healed after 5 months	Note: This patient had had a plastic operation to reform bladder at 3 years of age
24	1935	Gayet (*J. d'urol.*, 39: 295, 1935)	F	64	Adenocarcinoma	Refused treatment	Lost contact after 1 month
25	1935	Montpellier (*J. d'urol.*, 39: 493, 1935)	M	40	Squamous cell carcinoma	Case was studied, pyelogram made, split function; right ureter was dilated greatly; left ureter dilated	No operation; died a few days after admission; was febrile to 39–40°C. prior to death
26	1940	McCown, P. E. (*J. Urol.*, 43: 533, 1940)	M	62	Adenocarcinoma	Bilateral lumbar ureterostomy	Died; operation, shock; autopsy showed no metastases
27	1942	Graham, W. H. (*Brit. J. Surg.*, 30: 23–32, 1942)	M	48	Adenocarcinoma	Cystectomy; ureteral implant into wound margin	Home 8 months; did well; returned for sigmoid transplantation of ureters; good recovery
28	....	Graham, W. H., ibid.	M	53	Adenocarcinoma	Excision of extensive carcinomatosis bladder; ureters divided and left to form natural fistulas; repair of defect in abdominal wall; ureters discharge in a depression in the lower abdomen	Well 4 years after operation
29	1943	Abeshouse, B. S. (*J. Urol.*, 49: 259–289, 1943)	M	58	Adenocarcinoma	Hutchins and Hutchins method of ureteral transplantation	Died on 22nd postoperative day; acute bilateral pyelonephritis with azotemia
30	1945	Etherington-Wilson, W. (*Brit. J. Urol.*, 18: 62–64, 1945)	F	59	Adenocarcinoma	Ureteral transplantation; diathermy excision of malignant extrophy	Wound infection; died 7 weeks after operation
31	1946	Reid, Westcott, and Summers	M	63	Adenocarcinoma	Coffey I transplantation of ureters into sigmoid colon	Died on 11th postoperative day; pyelonephritis

placed in the pelvis of the right kidney. The non-protein nitrogen was 88 mg. per 100 cc. of blood. The patient showed moderate generalized edema but the next day seemed improved clinically. He continued to be irrational. However, on August 31, 1946, he seemed improved. He talked coherently, and the urine output was satisfactory. The non-protein nitrogen was 78 mg. per 100 cc. of blood. He expired at 5:30 A.M., September 1, 1946, on the eleventh postoperative day in an asthmatic attack that began five hours earlier.

The postmortem examination was limited to the abdomen. The anastomosis between the right ureter and the colon showed good healing whereas the left ureterocolonic anastomosis had

broken down. The left kidney, ureter and site of anastomosis to the colon were sent to the pathologist. The report on this tissue was:

"Chronic purulent pyelitis with very active ascending pyelonephritis. Multiple abscesses in the renal parenchyma. Large intestine shows purulent inflammation extending inward from the serosa and there is a necrotic and purulent tract passing through the wall. Nearby there are coarse sutures in position."

COMMENT

Exstrophy of the bladder is a rare congenital defect manifested by the absence of the anterior vesical and lower abdominal walls with eversion of the posterior bladder wall.

There are four types of exstrophy:[7] (1) Fissura vesicae superior in which there is a normal union of the pubis but a defect in the upper part of the bladder. (2) Fissura vesicae inferior in which the symphysis is developed normally, but the bladder is split inferiorly. (3) Typical complete exstrophy in which the symphysis pubis is absent with varying degrees of separation of the pubic bones. The everted bladder protrudes above the level of the surrounding abdominal wall. Urine may be seen spurting from the ureteral orifices; in older children the mucosa is inflamed. In the male epispadias is always present. The prostate may be absent or cryptorchidism may occur. At times a bifid scrotum may be observed. In the female the clitoris is cleft, the labia rudimentary and the urethra forms an open sulcus. A double vagina or a bicornate uterus may also be present. Other anomalies such as atresia ani hydrocephalus, spina bifida, cleft palate and umbilical hernia may exist in both sexes. (4) Exstrophy of the bladder complicated by intestinal openings on the extroverted area.

The umbilicus is located more caudad than usual or is absent.

According to Higgins[7] exstrophy of the bladder occurs once in 40,000 births and occurs seven times more frequently in the male. Of infants so afflicted, 50 per cent die before they reach the age of ten; 66 per cent

die before they reach twenty years of age. "The usual cause of death is the toxemia of renal infection or renal insufficiency resulting from a rapid progressive pyelonephritis or pyonephrosis.[1]

The present treatment of complete exstrophy of the bladder is to transplant the ureters into the rectosigmoid colon at an early age, to remove the exstrophic bladder and later correct the epispadiac. There is only one reported case of a complete exstrophy being successfully treated by plastic operations.[21]

In 1943, Higgins[7] presented nineteen cases of exstrophic bladder in which the patients were operated upon during the first year of life with only two postoperative deaths. His plan of operation is to transplant the right ureter into the rectosigmoid colon and after ten days to transplant the left ureter and excise the exstrophic bladder. The epispadias is corrected at five to six years of age. In two cases one ureter was transplanted and a nephrourectomy was performed on the opposite side because of a dilated ureter and hydronephrosis. His method of uretero-intestinal anastomosis is a modified Coffey 1 procedure.

In 1945, Schaefer[18] reported two cases of exstrophy of the bladder operated upon successfully at two months of age.

Attempts to anastomose the ureter to the colon were unsuccessful until Coffey[2,3,4] developed a method of uretero-intestinal anastomosis whereby the ureter lies submucosally for a few centimeters in the wall of the colon. As the fecal mass passed through the colon it would compress the ureter tending to prevent entrance of feces into it. In the Coffey 1 technic the severed ureters after being placed submucosally were introduced into the bowel through a small hole in the mucosa. In the Coffey 11 technic a ureteral catheter was sewed in the ureter and brought out through the anus; in the Coffey 111 technic the colon was not entered except to place a necrosing suture through it and the ureter. The Coffey technic of uretero-intestinal anas-

tomosis has carried a high mortality rate in the past.[6]

The problem has been to get a successful anastomosis without the development of obstruction and all the undesirable sequelae resulting therefrom.

Hinman and associates[7] from experimental work found that "Infection of the abdominal wound from contamination at the time of the operation is more imminent than peritonitis." The problem involved in transplanting the ureter into the colon is primarily to secure healing at the uretero-colonic anastomosis without the development of obstruction at this point. Hinman and associates[10,20] investigated the anatomy of the ureter as it entered the cloaca in birds, the cloaca being comparable to the colon in the potentiality of infection. They found that the ureteral musculature and the cloacal musculature in birds "retain a relationship as totally independent as if the ureter were artificially implanted." They found, however, that severance of the ureters in chickens and their reimplantation into the cloaca resulted in obstruction of the urinary tract in 91 per cent and ascending infection in 87 per cent of the cases. From this they concluded that: "The problems of ureterocloacal reimplantation, therefore, presents no peculiar features apart from those of uretero-intestinal anastomosis." They found further[19] that the reimplantation of the ureters into the bladder in dogs is not complicated by obstruction and ascending infection of the urinary tract which occurs after implantation of the ureters into the colon. From this they[11] concluded: "The end of the implanted ureter constitutes an area of crucial importance in determining the success or failure of a uretero-intestinal anastomosis."

Hutchins and Hutchins[12] reported two cases of exstrophy of the bladder in which the ureters were successfully transplanted into the rectum by an extraperitoneal type of operation. The ureteral orifice is transplanted with a cuff of vesical mucosa around it. This extraperitoneal approach

is now regarded as the method of choice in transplanting the ureters for exstrophy of the bladder.[1,15]

Jewett[13,14] has developed a two-stage type of uretero-intestinal anastomosis for cancer of the bladder, reporting thirty-three cases in 1944. Only one death has occurred in his last ten cases so operated upon.

Cancer occurring in the exstrophic bladder is usually adenocarcinoma whereas adenocarcinoma in the normally situated bladder is rare.[1,16] The treatment of exstrophy of the bladder should henceforth be more successful due to our present ability to control infection and to use of the extraperitoneal approach to the implantation of the ureters into the rectum. The operation should be done before the patient is six months of age.

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A. P. GRAHAM studied almost 200 cases of renal tumors and found that the initial symptom was hematuria alone in one-third of all patients and hematuria plus pain in over two-thirds of the patients. Retrograde pyelography was of diagnostic value in two-thirds of the subjects whereas an intravenous pyelogram was diagnostic in only one-sixth of the patients. When first seen, one-third of the patients already had metastatic lesions, principally to the lungs or bones, and almost one-half of the patients were inoperable. It would seem that hematuria is regarded by too many patients and doctors alike as probably signifying passage of a stone, or even as due to more trivial causes hence procrastination results and finally a "too late" diagnosis is made. Let us not forget hematuria *might* mean cancer somewhere in the genitourinary tract. (RICHARD A. LEONARDO, M.D.)

NEUROFIBROMA OF THE STOMACH

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VON RECKLINGHAUSEN[1] pointed out in his first paper dealing with neurofibromatosis of the skin that analogous tumor formations may occur in internal organs such as the gastrointestinal tract where they may attack either the serosa and subserosa or the submucosa. He pointed out that the tumor masses may grow outwardly producing nodular masses on the surface of the bowel wall, or may grow inwardly producing protrusions into the lumen of the gastrointestinal tract.

Askanazy[2] was the first to describe an isolated neurofibroma of the stomach; he suspected the myenteric plexus as the origin of the tumor because not only was there a marked increase in size of the plexus but also numerous ganglion cells between the nerve fibers were found.

Incidence. The incidence of neurofibroma of the stomach is extremely rare as borne out by the following statistical reports. During the period from 1907 to 1921, at the Mayo clinic[3] there were 195 operative procedures performed for gastric neoplasms. Of these, 168 were malignant and but twenty-seven were benign. During the period 1922 to 1927, at the Mayo Clinic[4] thirty-eight benign tumors of the stomach were reported, with the relative incidence of about one benign to eighty malignant, being the same as in the previous series. In both of these series there were no neurofibromas reported among the benign tumors.

Eliason and Wright in 1925[5] ·collected and analyzed 560 cases of benign tumors of the stomach and added fifty cases of their own. They made no mention of neurofibroma although eleven cases were listed as unclassified.

Judd and Hoerner,[6] in a report of fifty cases of benign tumors of the stomach, encountered one case of neurofibroma.

In a more recent study at the Lahey Clinic covering the five-year period ending 1943[7] there were 464 patients operated upon for gastric neoplasm. Of this group only nine, or 1.9 per cent, had benign growths. The benign lesions included leiomyoma, six, and one each of fibroadenoma, lipoma and neurofibroma.

Paul and Chapman[8] in 1945 reported that of 3,000 consecutive cases of gastric neoplasm at the Jefferson Hospital in Philadelphia, only thirteen were benign and but one was a neurofibroma. They also reported that of 1,500 consecutive gastroscopic examinations at the State University of Iowa Hospital only one case of neurofibroma was seen.

Finally, from the Cleveland Clinic, Root[9] reports in 1942 that out of 250,000 admission records, only seventeen cases of benign gastric tumors were encountered of which three cases were proven histologically to be neurofibroma.

It is seen that the foregoing reports, from the large clinics of various parts of the country, reveal the incidence of benign tumors of the stomach to range from less than ½ per cent to 2 per cent of all gastric neoplasms and that neurofibroma constitutes about 5 per cent of the benign lesions.

Pathology. Benign tumors of the stomach have been classified by Carli[10] in terms of their origin into four classes: (1) Those originating from epithelium include papilloma and adenoma; (2) those originating from connective tissue and vascular structure include lipoma, fibroma, myxoma and angioma; (3) those arising from muscular structures are myomas and (4) those arising from nerve structures are neuromas.

Those arising from the muscular coats in the stomach wall occur with the most frequency and comprise about 60 to 70 per cent of all benign tumors of the stomach. The neuromas, as previously stated, comprise but about 5 per cent.

Neurofibroma occurs more frequently on the lesser curvature of the stomach and more toward the pyloric end. They generally grow outward from the coats of the stomach producing a bulging mass into the peritoneal cavity. Occasionally, the growth points inward into the cavity of the stomach and in so doing thins out the protruding mucosa, and ulceration in the area of greatest tension soon follows. The tumors as a general rule are slow in their rate of growth. They do not infiltrate the surrounding tissue, the attachment being usually a localized type of dissecting growth in between the layers of stomach wall. This fact explains the ease with which the tumor may be surgically "shelled out," very little trauma being produced to the adjacent stomach tissue. Degeneration in the tumors of larger size is not uncommon. Most writers are of the opinion that about 10 per cent undergo malignant changes and these changes usually occur in those cases associated with a generalized neurofibromatosis.

Macroscopically, the tumors are usually of a pearly-grey color. The consistency is of a semi-hard character, appearing to be moderately soft at first palpation but then upon sustained pressure a definite hard firmness is appreciated. On section the surface is observed to be of a homogeneous, moist, glistening character and having a whitish-grey to pinkish-grey color. The mucous membrane and serous coats covering the tumor are usually smooth and glistening. The surface of the lesions are not very nodular as a rule.

Microscopic studies have caused many controversies among pathologists. They are all agreed that the majority of the tumors arise from the subserous or Auerbach's plexus and a lesser number arise from the submucous or Meissner's plexus.

The exact nervous elements from which growth takes place has been the point of disagreement. There is the group that holds that the proliferation takes origin from the cells of the nerve sheath (the cells of Schwann), and they have called the tumors Schwannomas and claims they are of ectodermal origin. There is another group that maintains that the tumors are of mesodermal origin, proliferation taking place from the perineurism and endoneurium. They claim the nerve sheath cells of Schwann take no part in the growth. They point out that the lesions have been observed to occur in nerves not having a sheath as the optic and olfactory nerves.[11] However, there are many writers[2] who are of the opinion that probably there is proliferation from both the mesodermal and the ectodermal elements except in cases of lesions involving cranial nerves.

Microscopically, the tumors characteristically all exhibit proliferation of nerve filaments arranged in thick bundles or tracts. Many fibroblastic cells intermingled in greater or lesser numbers with the fibrillar elements are seen. In many cases the dissecting character of the growth can be observed to split or dissect into the smooth muscle fibers. The nuclei in many areas are seen to be of a large oval shape and lay parallel to the fibrillar elements. Cystic degeneration is frequent and may be visible only on microscopic examination. Hemorrhages may occur in the softened tissue and myxoid degeneration has also been reported.

Symptomatology. · The symptoms of neurofibroma of the stomach are greatly variable, presenting no characteristic findings that might aid one in making a correct diagnosis. The symptoms depend a great deal upon the size and site of the lesion. The presence or absence of other coexistant disorders in the stomach[4] will greatly influence the clinical picture. The patient may present a typical ulcer story, with laboratory data lending support to the history given and in whom ulcer management will give temporary relief.[12] Hemor-

rhage is a frequent symptom which may be caused by the tumor itself or by a co-existant ulcer, the patient presenting hematemesis and/or tarry stools. The smaller tumors, not associated with ulceration of the mucosa and located distant from the pylorus, will present no symptoms and are only discovered at autopsy or at operation for coexistent disorders at a distant focus. If the tumor is situated close to the pylorus, signs and symptoms of obstruction are common. If the tumor is pedunculated in character, intussusception into the duodenum may occur, producing pain, nausea and vomiting. The pedunculated mass may recede and symptoms will subside. Repeated attacks of this nature would present an intermittent clinical picture of high obstruction.

Walters[13] states that the most frequent and most important sign of a benign tumor of the stomach is anemia. This anemia is caused by the so-called "weeping type" of bleeding and may escape the notice of a patient until pallor of the skin is marked or a blood examination reveals severe anemia to be present.

Pain of a severe character is an uncommon symptom unless, as pointed out, obstruction ensues with those lesions at the pylorus. Pain may be of a very dull nature and the sensation of heaviness in the stomach or a feeling of discomfort will be the only complaint. Vague symptoms of indigestion are a common finding.

Tumefaction in the epigastrium may be noticed for years, slowly, progressively increasing in size but with no distressing symptoms, especially in those cases in which the lesion is a distance from the pylorus. Large tumors of this nature, if close to the esophageal hiatus, may eventually cause symptoms of esophageal obstruction.

Loss of weight and strength may or may not be found, depending upon how much the appetite and the intake of food is impaired. As a general rule the appetite is good. Malignant degeneration, ascites,

perforation and peritonitis are of very rare occurrence.

Diagnosis. Because of the great preponderance of malignancy of the stomach over benign lesions, preoperative diagnosis of a benign lesion is rarely ever made. The possibility of a benign tumefaction should always be kept in mind for it is a source of courage to operate upon those patients who have symptoms that not only appear malignant clinically, but also in whom radiographic and laboratory findings seem to point to an absolute diagnosis of inoperable malignancy.

A careful, complete evaluation of the clinical signs and symptoms together with adequate laboratory study may in many cases enable us to arrive at a more correct diagnosis.

In twenty-three cases of histologically proven neurofibroma of the stomach,[14] ten cases were diagnosed as gastric malignancy, two cases as benign lesions of the stomach; in three instances a diagnosis of mesenteric neoplasm was made; in another three instances a diagnosis of ulcer was made and each of the remaining cases were diagnosed as a splenic or colonic tumor, a pararenal tumor, a retroperitoneal tumor and a biliary calculus respectively. This illustrates the interesting, but yet difficult problem that one is confronted with in making a diagnosis.

The following points toward making a differential diagnosis between benign and malignant tumors of the stomach are offered for consideration:

1. Neurofibroma of the stomach is occasionally associated with general neurofibromatosis. Sometimes, when they accompany cutaneous neurofibromatosis, there will be a history of hereditary tendency to the disease and a history of low mentality and arthritis deformans. The hereditary tendency has been definitely established.[15] Family history should be carefully investigated for evidence of neurofibromatosis and the possibility of a visceral localization must be seriously pondered in every case of multiple neurofibromatosis of the skin.

2. Spühler[14] has called attention to the fact that in reviewing twenty-three cases of solitary neurofibroma of the stomach that it is found in the more advanced age group, the youngest patient being fifty-nine years of age.

3. A history of hemorrhage with tarry stools on and off for an extended period of years would suggest a benign lesion rather than a malignant one.

4. The presence of a slow-growing tumor over a period of years without marked general signs of malignancy would be indicative of a benign growth.

5. A history of repeated intermittent attacks of high obstruction may be significant of a ball valve action or intussusception of the tumor mass into the duodenum, a fact more apt to occur with a benign lesion rather than a malignant one.

6. In tumors in which erosions into the mucosa may occur, fragments of tumor tissue may be found in the vomitus or be recovered from gastric lavage. This is of extremely rare occurrence but should it happen, biopsy would definitely establish the diagnosis.

7. Study of gastric analysis is of little aid in diagnosis for in most cases the curves are well within normal limits, there being very little change in gastric secretion.

8. Gastroscopic visualization together with peritoneoscopic visualization should prove of great assistance in establishing a diagnosis.

9. Regarding x-ray diagnosis of benign tumors of the stomach, Moore[16] states that a benign gastric tumor usually produces a filling defect which is centrally located and clearly circumscribed, sharp, smooth edged and punched out in appearance. The rugae may be distorted at the actual site of the lesion, but those surrounding the tumor are almost normal in their arrangement and distribution. The lesion will not interfere with peristalsis or obstruct the pylorus unless seated in the antrum. There will be no niche present or incisura seen because benign lesions do not cause contraction or spasm of the stomach. It must be conceded that while these signs are reasonably diagnostic they are not absolutely pathognomic of benignancy.

In view of the increase and diffusion of gastroscopic, peritoneoscopic and roentgenologic experience and the rather emphatic traits of benign tumors, it is probable that their diagnosis will be made more often and more confidently in the future than it has been in the past.

Treatment. Surgical intervention is the treatment of choice. Once a diagnosis of gastric neoplasm is made or suspected, all efforts toward the establishment of physiologic, protein, electrolyte and fluid balance are to be instituted. Hemorrhage, if present, and anemia are to be combatted.

When this preparation together with a careful survey of cardiac and general status of the patient is attained, then surgical exploration should be performed with the aim of removal and further study of the tumefaction.

Bearing in mind the multiplicity of neurofibromatosis, a careful search for other intra-abdominal conditions should be carried out at the time of operation. This point is well borne out by the case reported by Golm and Christeller[17] in which two tumors of the stomach were found, several in the small intestine, several on the periosteum of the tibia and one in the temporal lobe of the cerebrum.

The operative procedure itself depends upon the size and location of the lesion. If the lesion is not extensive and is situated on the lesser or greater curvature, simple excision followed by repair of the gastrotomy wound will suffice. If the lesion is at the pylorus, depending upon the amount of involvement, excision with gastroenterostomy or gastrectomy may be indicated. If the lesion is extensive and involves the greater and lesser curvatures at the mid-portion of the stomach, then a sleeve resection would be a wise choice of operation.

CASE REPORT

M. M., a white female sixty-five years of age, was admitted to the Norwegian Hospital on August 19, 1946, with a diagnosis of carcinoma

of the splenic flexure of the colon. The patient stated that in 1942 she first began to notice gas formation in her stomach shortly after eating. This progressed for a while and then she noticed that belching and nausea ensued soon after. She stated that at times it felt as if her stomach "fell" and then she would be greatly distressed until her stomach went back in place again. She had many periods, however, when she was entirely free of symptoms for weeks at a time. Her periods of distress lasted sometimes for days and occasionally she would be distressed to a greater or lesser degree for a week. For the past four months, however, she stated that an annoying, dull ache has been more or less constantly present in the left upper part of her abdomen. The dull ache was usually aggravated by the intake of any and all foods and was not relieved by any food or stomach medicine. She suffered a great deal with gaseous distention, eructations and nausea and almost invariably had to have a defecation after each meal. Of late she has also noticed alternating loose and hard stools, but the color has always been normal and no blood or mucus was ever noticed. There has been a declining appetite to the extent that the odor of food is unpleasant. Her best weight has never been more than 116 pounds and at present it is 107 pounds. Physical adequacy has also been failing. The review of the systems revealed no other complaints. Her past history revealed a hysterectomy for fibroids and an appendectomy in 1936; otherwise her health had always been good with the exception of an occasional cold. The family history was non-contributory.

Physical examination revealed an elderly, white female of short height and medium build. Nutrition appeared good although she weighed only 107 pounds. Hemic component appeared fair. She was admitted ambulatory and was in no distress but her general physical stamina did not appear good. Examination was essentially normal throughout, with the exception of the abdomen. The abdomen was flat and soft. There was a palpable, discrete, firm, somewhat irregular, slightly tender mass in the left subcostrum which moved up and down on deep respiration. The mass was about 5 by 5 by 8 cm. in size.

The admission blood study revealed 3,240,-000 red blood cells, 60 per cent hemoglobin, 19,950 white blood cells with 87 per cent polymorphonuclear cells and 13 per cent lymphocytic cells. The urine examination re-
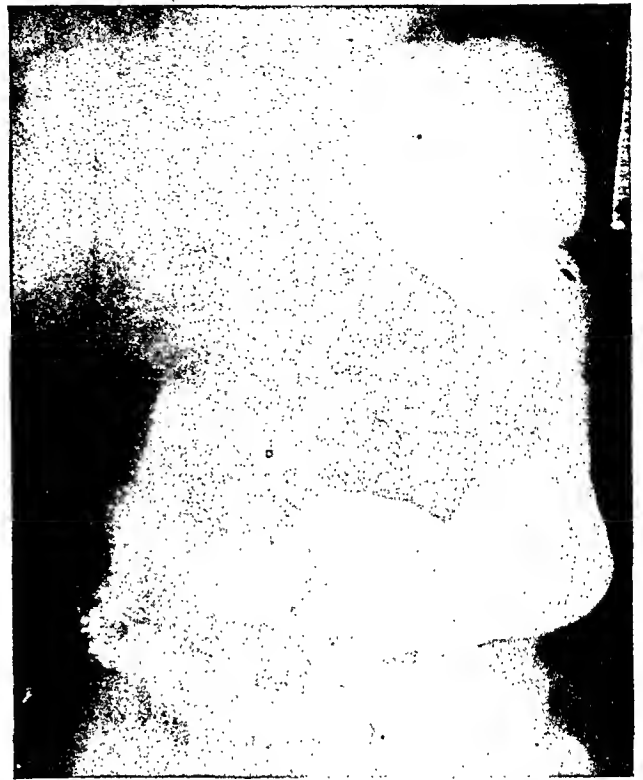

FIG. 1. Gastrointestinal series; five-hour film.

vealed a trace of albumin and some granular débris with a few epithelial cells microscopically. Blood chemistry studies were within a normal range and gastric analysis was also within normal limits. Barium enema studies revealed a redundancy of the transverse, descending and sigmoid colon with a partial loss of haustral markings of the descending and sigmoid colon. The postevacuation film showed that practically all the barium had been expelled.

Study of the gastrointestinal series revealed a large, well rounded filling defect on the lesser curvature of the stomach. (Fig. 1.) A few irregular collections of barium appeared in this area which apparently communicated with another irregular collection of barium about 5 to 6 cm. medially and was situated just to the left of the second lumbar vertebra. This second collection of barium was present both in the five-hour and twenty-four hour films but was not seen in the forty-eight-hour film. The meal proceeded normally throughout the balance of the gastrointestinal tract and no other pathologic condition was noted.

The patient was given adequate physiologic preparation with 500 cc. of whole blood, intravenous glucose, saline, vitamins and proteins; and on August 30th, eleven days after admission, an operation was performed.

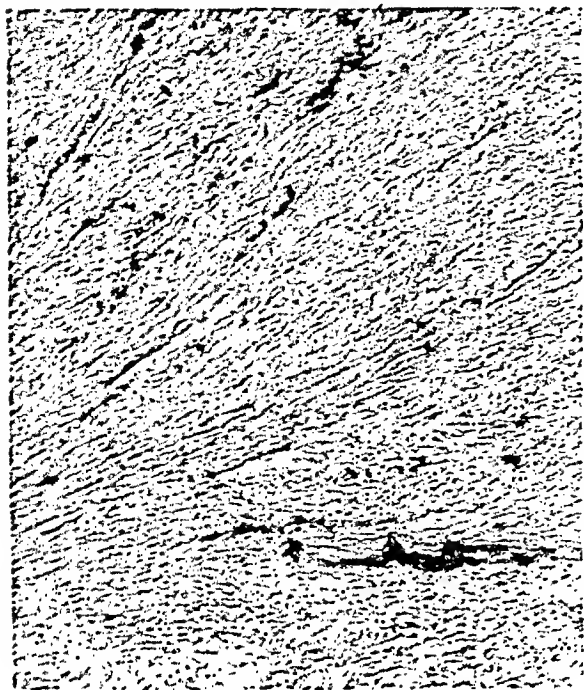A left rectus incision about 10 cm. long was

FIG. 2. Microphotograph; magnification × 130.

employed. The stomach was found to be the site of a neoplastic involvement of the lesser curvature measuring about 6 by 6 by 8 cm. The mass was of a pale, whitish color, semi-hard and rubberish in consistency. There were several small whirl-like masses about 1 to 1.5 cm. in diameter attached to the periphery of the mass. The mass was easily enucleated from between the layers of the stomach wall and its connection with the stomach was severed. The gastrotomy wound so formed was closed with an atraumatic chromic No. 00 Connell suture and reinforced with another layer of continuous mattress, seromuscular, fine, black silk suture.

The center of the mass was necrotic and contained mucopurulent material of a very foul odor. The cut surface revealed many areas of hemorrhage and degeneration. The tumor mass was situated between the seromuscular and the submucous layers of the stomach wall. At one point in the inferior pole of the mass there was a communication with the lumen of the stomach which admitted about two fingers.

The microscopic report revealed a thick, hyalinized, fibrous connective tissue capsule. Beneath the capsule were many interlacing strands of fairly dense fibrous connective tissue supporting pale, vesicular spindle-shaped cells. Many bundles or tracts of nerve filament proliferation were seen (Fig. 2.) There were many areas of myxomatous degeneration throughout together with occasional collections of round cells. Pathologic diagnosis: Neurofibroma.

The patient was given adequate physiologic support postoperatively, whole blood, 500 cc., glucose, saline, proteins and vitamins intravenously, for two days. Then small, frequent feedings by mouth for two days were also given. These were gradually increased in amount and intravenous aid gradually decreased. After ten days intravenous aid was entirely discontinued and patient continued to make steady progress. On the twenty-second day postoperatively the patient, having been on a full, generous diet with no limitation for five days, was discharged as fully recovered.

She has been seen several times and her general condition has steadily improved. She is doing all her usual housework, has gained in weight and vigor. She does admit to an occasional "bit of gas," but claims she takes no stomach medicine for relief because the "bit of gas" is quickly belched up and she feels fine. She experiences none of her previous distress or feeling that her stomach is "falling." She is eating well with absolutely no restrictions of any particular items of food.

SUMMARY

1. A general review of neurofibroma of the stomach is presented.

2. Certain points deemed helpful in differentiating between malignancy and benignancy of gastric neoplasm are offered for consideration.

3. A plea is made to make every effort to bring patients with gastric neoplasms within physiologic balance so that they may be explored, and the occasional patient with benign lesion may be saved and not be classified as inoperable and allowed to suffer.

4. A case of solitary neurofibroma of the stomach is reported.

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H. K. RANSOM reports his studies on sixty cases of total gastrectomy in which the operative mortality was close to 25 per cent. The author is convinced that such patients lead a reasonably comfortable life and that the incidence of postoperative pernicious anemia is low, only two patients developing this complication some five years after total gastrectomy. (RICHARD A. LEONARDO, M.D.)

PRIMARY HEMANGIOMA OF MUSCLE

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HEMANGIOMA of skeletal muscle is a relatively rare disease. In a series of 1,308 hemangiomas, Watson and McCarthy reported that ten, or 0.8 per cent, occurred in skeletal muscle. Since the first reported hemangioma of skeletal muscle in 1843, some 353 cases have been reported. Shallow, Eger and Wagner in a recent article presented a comprehensive survey of primary hemangiomatous tumors of skeletal muscle, with a complete analytical review of the literature to date, and added two cases of their own.

The following case report is of interest because the hemangiomatous tumor involved the entire external and internal abdominal oblique muscles of the right abdominal wall and hemangioma of muscle was considered in the differential diagnosis because of the presence of phleboliths

CASE REPORT

A white, twenty-eight year old soldier, was admitted to this hospital June 22, 1945, because of a mass in his right lower quadrant. He stated that in 1936 while working for the CCC camps he was struck in the right lower abdomen by a stump. That evening he was taken to a hospital where he was told that a "blood-clot" necessitated an operation. He was asked if he wanted his appendix removed at the same time and consented. On returning to duty about three months later, he noticed a "throbbing" and soreness in his right lower quadrant just above the incision. For two years prior to induction he worked as a ship fitter and as a stockman in a wholesale house. All during this time he was aware of a mass in the right lower abdomen. As he described it, the mass was a hard, full area, which became sore on exertion. The patient had been in the army twelve weeks and had finished basic training two weeks before admission to this hospital. He said that during the past four weeks the

"lump" had become larger and more tender. The family history was non-contributory.

Physical examination disclosed a high, well-healed, McBurney incision in the right lower quadrant. Beneath the upper portion of this scar was a firm, fusiform, fixed mass extending laterally to the ilium and upward to a point about 4 cm. above the anterior superior spine. The only clear-cut margin of the mass was the upper one which was rounded and smooth. The mass measured approximately 12 by 8 cm. Palpation gave the impression of rubbery consistency and compressibility. The mass and also the adjacent iliac bone were moderately tender. The mass was dull to percussion. Elsewhere the abdomen was soft and non-tender; no other masses were palpable; otherwise, the physical examination was negative.

The blood count, urinalysis, blood Kahn, intravenous pyelogram, barium enema and stool examinations were reported as normal.

Roentgenographic studies of the chest and soft tissue films of the abdominal wall in the region of the tumor were made. The chest was normal. The abdomen showed a soft tissue mass, seen in contour. It was not particularly homogeneous; rather, it had a lumpy appearance. No definite capsule or limiting membrane was seen. The bone of the right ilium was intact and did not appear to be involved. Superimposed upon the liver region were at least three small calcified bodies which had the appearance of phleboliths.

It was our impression that the mass was extra-abdominal and was a fibrosarcoma or lipoma. Because the x-ray studies revealed calcified bodies in the soft tissue mass, hemangioma of muscle was also suspected. On the basis of a palpable mass within the right anterolateral abdominal wall, surgical exploration was undertaken. Under spinal anesthesia, an incision was made over the mass in its long axis. Skin flaps were dissected laterally and medially, exposing a large fusiform tumor which extended from the right twelfth rib to the inguinal ligament, laterally to the crest of the ilium and lumbodorsal area and medially to

FIG. 1. Cut surfaces of muscle segments after fixation in formalin. Vascular channels of varying caliber permeate the muscles. (Photography by U. S. Army Corps.)

within 2 cm. of the lateral border of the rectus sheath. The tumor mass infiltrated the muscular portions of the entire external and internal abdominal oblique muscles. No cleavage plane could be made out between these two muscles, both being fused by the infiltrating tumor. In depth, the tumor attained the transversalis fascia but did not involve it. It did not extend beyond the superficial fascia. A piece of tissue, removed for frozen section, showed hemangioma of muscle. Although the tumor was not histologically malignant, the clinical history and behavior of this lesion justified wide excision of all involved tissue. The entire external and internal abdominal oblique muscles were excised. Blood loss from the vascular tumor necessitated transfusion on the table. However, the patient's convalescence was uneventful.

The specimen consisted of many large pieces of voluntary muscle; the total weight was approximately 1,200 Gm. (Fig. 1.) Although they varied in size and shape, they were structurally similar. Large areas of muscle fibers appeared normal, but about half of each cross-section was composed of vascular channels, fibrous connective tissue and yellow fat. The channels varied in diameter and all had thin walls. There were occasional hemorrhages into the surrounding tissues. The thin-walled vessels were located in the muscular tissue as well as in the perimysial and intermuscular fascial planes. Thus was formed a solid, firm, rubbery mass.

Isolated clusters of muscle fibers appeared tan and dull in contrast to the red, glistening more normal fibers. Many slides from representative pieces of the specimen were studied. (Figs. 2 and 3.) The essential lesion in all was similar. Blood-filled spaces of large caliber were lined by a single layer of endothelium. These vessels permeated the striated muscular tissue, accompanied by varying degrees of fibrous scarring and fatty replacement. Often the fibrous areas contained infiltrates of lymphocytes, plasma cells and eosinophil polymorphonuclear leukocytes. Scattered deposits of greyish-brown, finely granular pigment were seen in the interstices of the fibrous tissue. Muscle fibers isolated by the process appear pale and swollen. No cytologic evidence of malignancy was noted in any of the various types of cells. Pathological Diagnosis: Hemangioma, cavernous, of muscle.

The patient was observed at regular intervals. On December 10, 1945, six months after operation, he appeared in excellent health. The wound was well healed and there was no evidence of recurrence of the tumor or development of a hernia.

The clinical picture of hemangioma of muscle is varied, depending upon the site of occurrence. Symptoms may persist from one to several years. Pain, the most com-
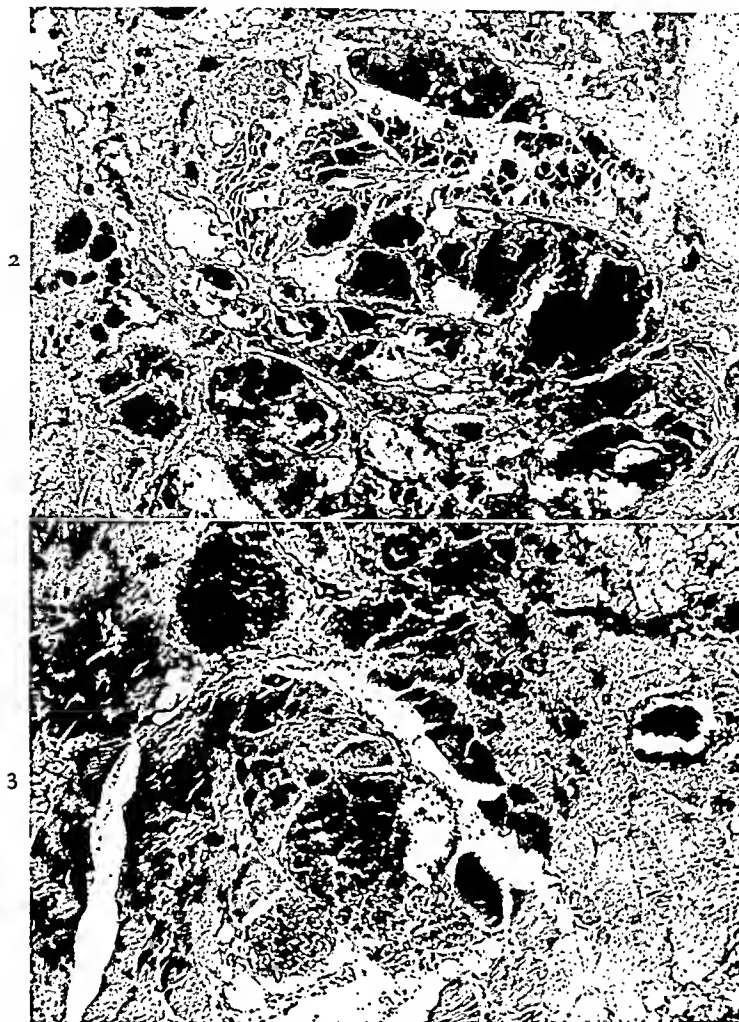
FIG. 2. Microscopic picture of cavernous hemangioma showing fatty and fibrous replacement of degenerating and atrophic muscle fibers. × 30. (Photography by Army Medical Museum.)

FIG. 3. Angiomatous invasion of muscle bundles, interstitial round cell infiltration, fatty and fibrous replacement. × 145. (Photography by Army Medical Museum.)

mon symptom, is localized at the site of the tumor. It varies from a dull aching fullness to a sharp throbbing sensation. The pain may be aggravated by exertion and will usually subside with rest. Palpation usually reveals a fixed mass which may be ill defined or sharply demarcated. The mass may feel fluctuant, soft or firm; more often it has a rubbery consistency. It is usually tender to deep pressure and pulsations may be detected. The most common site of occurrence is in the muscles of the lower extremities although any striated muscle may be affected. One or several muscles may be involved.

COMMENT

It is interesting to speculate upon the possibility of the existence of the tumor prior to injury. The concensus is that hemangiomas are congenital and that trauma is frequently an important factor in activating the growth of the tumor and development of symptoms. In the collected series of Shallow, Eger and Wagner, trauma was reported related to the onset of symptoms in 17 per cent of the cases.

The condition must be differentiated from hematoma, fibroma, neurofibroma, fibrosarcoma, lipoma, hydatid cyst, myoblastoma and rhabdomyoma. Exploratory

puncture and roentgenological studies are the most valuable aids in establishing a correct diagnosis. Phleboliths shown by x-ray films are present in about 48 per cent of the cases. Exploratory puncture reveals blood in approximately 98 per cent of the cases.

Treatment. Surgical excision is the procedure of choice. Shallow, Eger and Wagner report improvement or cures in 90 per cent of their collected cases.

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Bony defects are present, especially in the skull or ribs. When removed and examined, the tissue is found to resemble that present in cases of Schüller-Christian's disease. However, from then on, no further similarity has been found between these two conditions. Patients with eosinophilic granuloma of bone, present localized pain and tenderness and later a localized swelling, first hard and later softer. Slight fever and leukocytosis may be present but eosinophilia is a rather rare finding, occurring in only 10 per cent of the cases. Treatment is by surgery and radiation and the condition is apparently not fatal. (RICHARD A. LEONARDO, M.D.)

UNRUPTURED PRIMARY OVARIAN PREGNANCY*

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THE possibility of ovarian pregnancy has not always been admitted. Lawson Tait,[1] the first surgeon to operate on a patient with tubal pregnancy, declared that ovarian pregnancy was an impossibility. Authorities report that many of the early cases were those of dermoid cysts.[2] However, Catherine van Tussenbroek[3] in 1893 and Thompson[4] nine years later, produced undeniable proof of the existence of ovarian pregnancy.

In 1941, Wilson and Robins[5] reviewed the literature on ovarian pregnancy and stated that, including their own case, there were fifty-nine authentic cases reported. This review included the forty-eight undeniable cases reported by Wollner[6] in 1932, the four cases reviewed by Russell and Black[7] in 1940 and seven more reported since 1940. Thomas[8] reported a case in 1943, and stated that five more cases had been published between June, 1941 and November, 1941 by Simard,[9] Nicholls,[10] Eckerson,[11] Ross and Gledhill[12] and Curtis,[13] bringing the total to sixty-five. A review of American and British journals to date, since the report of Thomas, reveals fifteen additional cases, including the one of Mann, Meranze and Lief[15] who argued the secondary or primary nature of their case.[14—27] It is assumed that the case reported herein brings the total to eighty-one.

It is worthy of note that in 1942, Courtiss[16] stated in his review of the literature that there were eighty-nine authentic cases reported, including his own, Wollner's[48] undeniable cases and others. Therefore, it is apparent that there is a great discrepancy as to the number of authentic cases published. This confusion among authorities apparently results from the lack of strict interpretation of primary ovarian pregnancy according to the definite criteria set forth by Spiegelberg[28] in 1878. Although the actual incidence of the condition remains uncertain, there is no doubt that primary ovarian pregnancy is still a comparative rarity.

CASE REPORT

The patient, Mrs. L. G., a white, married housewife, aged twenty-three, was first seen in the office by one of us (S. S. W.) on July 8, 1946. She complained of pain in the right lower quadrant, irregular menstrual bleeding, dyspareunia and dyschezia. Two weeks after her last menstrual period, on June 1, 1946, she began to have vaginal spotting. This staining continued for one week and then on June 26, 1946, she started to bleed moderately; this continued up until the time of the examination. For the past month she had noticed mild dyspareunia and dyschezia. For several weeks she experienced pain and fullness in the breasts. Her menstrual and marital history revealed that the menarche occurred at twelve years of age and her periods had always been regular of the 5/28 day type with slight dysmenorrhea on the first day. The patient has been married four years during most of which her husband had been in military service. He has been back eight months. She has never previously been pregnant. The past history disclosed that her only serious illness had been scarlet fever. Her surgical history consisted of tonsillectomy and adenoidectomy at the age of seventeen and an appendectomy in 1941.

The physical examination disclosed the following: The patient did not appear acutely ill; she weighed 119.5 pounds and was 63 inches in height. Her blood pressure was 90/60. The general physical examination was essentially negative. The abdominal examination revealed a well healed McBurney incision and some tenderness low in the right lower quadrant. The pelvic examination demonstrated a nulliparous marital outlet. There was no evidence of urethritis, skenitis or bartholinitis. The cervix was in the axis of the vagina, was normal

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in consistency and freely moveable. The uterus
was normal in size and anteverted. Mobility of
the uterus caused only slight pain. In the right
adnexal region, there was a slightly tender,
doughy mass about the size of an egg. The left
adnexa was slightly tender and thickened. The
clinical impression was that she had chronic
pelvic inflammatory disease and she was put to
bed and given sulfadiazine 1 Gm. four times
a day for five days.

On July 15, 1946, the patient still had vaginal
bleeding but complained of less pain in her
right lower quadrant. A pelvic examination at
this time revealed a normal sized anteverted
uterus. The cervix was normal in consistency.
The left adenexa was not palpable or tender.
In the right adnexal region, there was a slightly
tender, doughy mass the size of a small orange.
The sedimentation rate was 23 mm./hr. The
tentative diagnosis at this time was that of a
corpus luteum cyst and an operation was
advised.

On July 24, 1946, under nitrous oxide and
ether anesthesia, the abdomen was entered
through a Pfannenstiel incision. There was no
free blood in the peritoneal cavity. An examin-
ation of the pelvis revealed a normal sized
uterus in second degree retroversion. The left
tube and ovary were normal. The right ovary,
except for one pole, was replaced by a hemor-
rhagic, discrete, doughy, cystic mass which was
attached by a few, fine adhesions to the pos-
terior surface of the uterus and right broad
ligament. The adhesions were easily separated
by blunt dissection and the cystic mass, which
was considered to be a corpus luteum cyst in
spite of its unusually large size, was brought
into the wound. The right tube was normal in
appearance and completely free from the
ovarian mass. The fimbriated end of the tube,
which was patent, was not attached to the
ovarian mass. This right tube, being normal,
was not removed. The mesovarium was grasped
just below the ovarian mass by two curved
forceps, one applied on the utero-ovarian liga-
ment and the other meeting it on the infundi-
bulopelvic ligament. A right oophorectomy was
performed by excision of the mass above the
clamps. The mesovarium was then coapted
with continuous chronic catgut suture. The
uterus was suspended by attaching the round
ligaments by mattress sutures to the anterior
surface of the uterus. The abdomen was closed
in layers. The postoperative course of the

FIG. 1. Gross specimen sectioned to demonstrate
the gestational sac, surrounding blood and
ovarian tissue, with retention cysts exhibited in
one pole of the ovary.

patient was completely uneventful and she
was discharged from the hospital on the tenth
postoperative day.

The pathologic report by Dr. Clarence I.
Owen, pathologist, Grace Hospital was as
follows: Gross: The specimen was an ovoid
shaped mass measuring 7 by 6 by 5 cm. in size.
The surface was smooth and nodulated. On
cross section it was found to be an ovary with
a centrally located cyst which was lined by a
glistening, smooth, sac-like membrane except
for one area where there was a slight elevation.
Surrounding the lining, there was a considerable
amount of blood clot intermingled with some-
what friable tissue and, on the outside of this,
there was an outer continuous layer of ovarian
tissue. (Fig. 1.) The ovarian tissue could be
recognized as forming a cap at one pole meas-
uring 3 by 3 cm. in size and from 8 to 15 mm.
in thickness. Within the ovarian tissue there
were a few retention cysts. On the side opposite
to the ovarian cap the wall of the cyst appeared

FIG. 2. Photomicrograph of blood surrounding gestational sac demonstrating a clump of villi (high power magnification × 100).

to be stretched and quite thin. There was no rupture or break in continuity of the sac at any point. There was no demonstrable fetus within the sac.

Microscopically, ovarian tissue was recognized in the areas described grossly. The cystic structure was found to be a pregnancy in which the fetus was not present. The sac-like membrane consisted of amnion and chorion and surrounding this there was a layer of blood clot within the meshes of which numerous chorionic villi were present. The villi showed both a Langhans' and syncytial layer. (Fig. 2.) The external layer consisted of ovarian stroma and at one pole several retention cysts were seen. Lutein cells, arranged in long sheets (not in the usual manner), were identified in one portion of the ovary.

Diagnosis: Unruptured ovarian pregnancy.

COMMENTS

That the case reported is a true unruptured primary ovarian pregnancy is evidenced by the operative findings and the gross and microscopic examination of the specimen removed at operation. Spiegelberg's criteria are strictly fulfilled, namely: (1) that the fallopian tube on the affected side, including the fimbriae ovarica, be intact and the former completely separated from the ovary; (2) that the gestation sac occupy the position of the ovary; (3) that the sac be connected with the uterus by the ovarian ligament and (4) that ovarian tissue be demonstrated in the wall of the sac.

It is necessary to distinguish at this time between primary and secondary ovarian pregnancy. In the primary type, the fertilized ovum undergoes its entire development within the ovary. In the secondary type, the fertilized ovum begins its development outside of the ovary in some other structure, usually the tube, eventually becomes separated from that structure and then attaches itself to the ovary and continues its development there.

The basis for our classification of this case as one of the primary type, is a total absence, on gross and microscopic examination, of any tissue foreign to the ovary except for the gestational elements themselves.

Recently, Curtis,[13] reporting a case of his own as a primary ovarian pregnancy, casts doubt on that diagnosis by describing a "toboggan slide" position of the fimbriae. He theorizes that the ovum did not necessarily have to be fertilized within the follicle but could be fertilized in the tube and, then secondarily, slide back again on to the ovary.

MECHANISM OF OVARIAN PREGNANCY

Implantation and development of the fertilized ovum may take place ectopically in the ovary, (1) well within its substance, i.e., where the gestation sac forms in the depths of the ovary, and is evenly covered by ovarian tissue. This mode of implantation can be of two types, viz., intrafollicular or interstitial and (2) on the periphery where the fetal sac grows into the peritoneal cavity. This method of ovarian implantation may likewise be of two types, viz., superficial or suprafollicular, as postulated by Teacher,[29] Greenhill[30] and Litzenberg.[31]

These four modes of ovarian implantation are briefly described as follows:

Intrafollicular Ovarian Implantation. According to Leopold,[32] primary ovarian pregnancy always results from fertilization of the ovum before it escapes the graafian

follicle. Until recently, this was considered to be the only mechanism by which ovarian pregnancy could occur. The rarity of ovarian pregnancy, in spite of the accessibility of the ovary and its ruptured follicles to the spermatozoon, can be explained by the fact that the ovum, as it exists in the ovary, must undergo some degree of maturation before it is capable of becoming fertilized. This normally occurs only during its passage through the tube.

Anything that interferes with the expulsion of the ovum from the ruptured follicle may permit a sufficient passage of time to allow the ovum to mature and be capable of becoming fertilized. According to Leopold, the escape of the ovum from the follicle may be retarded by a tortuous channel of exit or by a deep follicle rupturing and discharging its ovum into the cavity of a more superficial one, the spermatozoon reaching the mature ovum after rupture of the superficial follicle. Another theory is that the expulsion of the ovum is retarded by a small blood clot at the site of the rupture; still another theory, as championed by Wollner,[6] is that the ovum is not extruded due to ovarian pathologic conditions involving the tunica albuginea. If the force of the expulsion of the ovum is dependent upon the balance between intrafollicular pressure and the resistance of the surrounding tissue, it is reasonable to assume that any pathologic change in either of the two factors will produce a faulty ovulation.

All of these theories account for the centrally located cases of ovarian pregnancy. Hewetson and Lloyd[33] suggest one more mechanism, i.e., that the fertilized ovum burrows more deeply into the ovary by virtue of its cytolytic power.

In our case, the presence of lutein cells indicates the strong possibility that the ovum, if not fertilized in the follicle, was implanted there after fertilization and then eroded its way deeper into the ovarian tissue.

Interstitial or Juxtafollicular Ovarian

Implantation. The implantation occurs outside the follicle in the stroma of the ovary. The theoretical explanation here is that the follicle bursts laterally, due to a tough capsule preventing the normal outward release of the ovum which escapes into the interstitial portion of the ovary. Here it is found by the spermatozoon which enters the follicle when it finally bursts outward. King[34] reports such an authentic interstitial ovarian pregnancy.

Superficial Ovarian Implantation. Here nidation takes place on the surface of the ovary as a result of pathologic or anatomic changes, e.g., peritoneal adhesions, periooophoritis, thickening of the tunica albuginea, cystic degeneration or endometriosis and also abnormal furrows, wrinkles or sulci.

Novak[35] agrees with Meyer that the most common mechanism is cortical implantation of the egg. The theory, advanced by Webster[36] in 1904 and Sutton[37] in 1924, that implantation cannot occur except in the presence of endometrium or displaced müllerian tissue in the ovary is now generally rejected. Although McKenzie,[19] Curtis[13] etc. report cases of ovarian pregnancy associated with the presence of endometrial tissue, there are others, Haeubner,[38] Jordan[39] the authors, who are reporting cases without any detectable endometrial tissue. Stux[40] believes the report of Brougha and Robinson and his own case prove the occurrence of superficial ovarian pregnancy.

In some of the cases of superficial ovarian pregnancy, the corpus lutem was in the opposite ovary and it is likely that during transmigration the ovum had sufficient time to reach the morphoblastic stage when it could more easily attach itself to the surface of the ovary.

Suprafollicular Implantation. This is another type of superficial implantation. It is caused by imprisonment of the ovum by a superficial blood clot at the opening of the burst follicle. The ovum is thus held at its exit where it is fertilized, becomes implanted and develops.

This mode of implantation is exemplified by the case reported by Gerstel.[41] The only other similar case reported in the literature is by Seedorf.[42]

In spite of our present knowledge of ovarian implantation, there is still much difficulty, in many cases, in establishing the particular method of implantation.

Course and Termination of Ovarian Pregnancy. Ovarian pregnancy, like tubal, is pathologic from the beginning; the ovary, like the tube, being anatomically unfit for this purpose. The early implantation and placentation do not differ greatly from intra-uterine pregnancy except for the absence of decidua or decidual cells.

Jordan,[39] who made a careful histologic study of these masses of cells which are usually interpreted as decidual, considers them as probably being lutein cells; these were so interpreted in our specimen.

The placenta usually consists of ovarian tissue, fibrin from the excessive hemorrhage, lutein cells and fetal and chorionic elements, trophoblast and villi.

The usual course in ovarian pregnancy is early rupture of the ovary with the ovum completely or partly extruded into the peritoneal cavity (ruptured ovarian pregnancy). This occurs in the majority of cases by the end of the first trimester, accompanied by moderate or profuse intra-abdominal hemorrhage. In a number of cases, the ovum may die early and be completely absorbed (this probably occurred in our patient); in other cases, the products of conception may undergo degenerative changes at an early period without rupture and give rise to a tumor of varying size (unruptured ovarian pregnancy). This tumor usually consists of a capsule of ovarian tissue enclosing a mass of blood and chorionic villi. It may or may not contain an amniotic cavity.

Apparently, the ovary can accommodate itself to the growing pregnancy more readily than the tube and a higher percentage of recorded cases go to term. Thus, Nichols,[10] in reporting a case of ovarian pregnancy with living mother and child,

states that the literature contained thirty-eight cases of ovarian pregnancy which went to the age of viability (seven months) or beyond. These cases cannot be differentiated clinically from abdominal pregnancies.

Another possible termination, as with tubal pregnancy, is the formation of a lithopedion which may be carried for years. In some cases, hydatiform mole formation has been observed.[30] Another interesting observation has been the occurrence of eclampsia in ovarian pregnancy.[17]

Differential Diagnosis. The differential diagnosis depends upon the signs and symptoms elicited preoperatively.

In the presence of intra-abdominal hemorrhage, a differential diagnosis from ectopic tubal gestation cannot be made, positively, before operation. This fact is borne out in the literature when the majority of such cases are diagnosed as ruptured tubal pregnancies. Other causes of ovarian hemorrhage enumerated by Traugott[43] are leukemia, phosphorous poisoning, hemorrhagic diathesis, hypertension, neoplasms, cysts with acute torsion, ruptured graafian follicles, endometriosis and corpus luteum cysts.

In the cases of unruptured tubal pregnancy, when the involved ovary remains intact and forms a cystic tumor, the preoperative diagnosis, as in our case and several such cases in the literature, was corpus luteum cyst.

A corpus luteum cyst often presents no associated characteristic symptoms. With large corpus luteum cysts, however, the symptoms may fall into a certain pattern such as delay of menstruation, slight vaginal bleeding and pain on the involved side. These symptoms, associated with a palpable cyst-like mass, are very suggestive of a diagnosis of corpus luteum cyst. Even at operation, especially if the cyst be intact, a large corpus luteum cyst may be confused with an unruptured primary ovarian pregnancy.

In recent years, particularly in this country, the number of ovarian pregnancies reported has become accelerated (two or

three per year). This is due, we are convinced, to a more stringent examination of the diseased tissues at operation and in the pathologic laboratory. The exact diagnosis in many cases remains obscure until the pathologic specimen is carefully studied.

We strongly recommend that in all cases when a hematoma of any size is found in the ovary, that the possibility of an early ovarian pregnancy be considered and a careful search be made for chorionic villi or parts of a fetus in it.

SUMMARY

1. A case of unruptured primary ovarian pregnancy is presented.

2. The recent English literature is reviewed.

3. The various mechanisms of implantation of primary ovarian pregnancy are described.

4. The course, termination and the differential diagnosis of primary ovarian pregnancy are discussed.

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EVENTRATION OF DIAPHRAGM

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THE condition known as eventration of the diaphragm is not really a diaphragmatic hernia but a diffuse relaxation of half of the diaphragm, simulating a hernia. The exact cause of the condition is not known and the degree of eventration varies greatly.

Eventration of the diaphragm may be congenital or acquired. If the normal development of the phrenic nerve is disturbed, it may be of congenital origin. The muscle layer is replaced by fatty and fibrous tissue and the affected leaf rises into the thorax, followed by some of the abdominal contents. The condition is more common on the left side. Incarceration of the viscera is not possible because there is nothing to correspond to the neck of a hernial sac. Acquired eventration may be acute or chronic. It may follow a lesion of the phrenic nerve or it may be due to degenerative changes in the musculature of the diaphragm itself. Acute eventration of the diaphragm may follow one of the infectious diseases, run a course of a few weeks and disappear.

CHRONIC EVENTRATION OF THE DIAPHRAGM

Under this heading would come the remainder of the cases in which the diaphragm is permanently disabled. This condition may exist some time without symptoms. In all marked cases dyspnea, cyanosis and palpitation are present on the slightest exertion due to the pressure of the abdominal organs on the heart and lungs; difficulty is also encountered in lying upon the healthy side. Pain is an inconstant symptom; constipation is usual and dysphagia paradoxa and hematemesis are occasional. The heart usually is displaced to the right as the lesions are most frequent on the left side. Over the affected side the chest is tympanitic; ausculation reveals gurgling bowel sounds and the absence of breath sounds.

The diagnosis is very difficult, even in the most extreme cases, without the aid of the x-ray. With x-ray ordinarily it is not difficult to distinguish hernia from eventration of the diaphragm. In the latter there is a limited, paradoxical respiratory movement of the diaphragm which is high and well outlined; the abdominal contents lie below. With hernia, on the other hand, the torn diaphragm is not easily outlined and only a portion of the leaflet may be seen. This is not elevated nor is paradoxical movement present but the herniated stomach, colon or small bowel can be seen protruding through the diaphragmatic rent in the thorax. Cases arise, however, in which these distinguishing features are lacking and the exact diagnosis is rendered uncertain.

CASE REPORT

J. J., a white male, age sixty-nine, was admitted to the hospital May 28, 1946, complaining of dyspnea on slight exertion, cyanosis, palpitation, a dull ache over the heart which occasionally was sharp and radiated to the right arm, belching, flatulence, constipation, occasional difficulty in swallowing and severe cramping pains in the epigastrum and lower chest after eating. He had lost weight 30 pounds during the past year which he attrib-
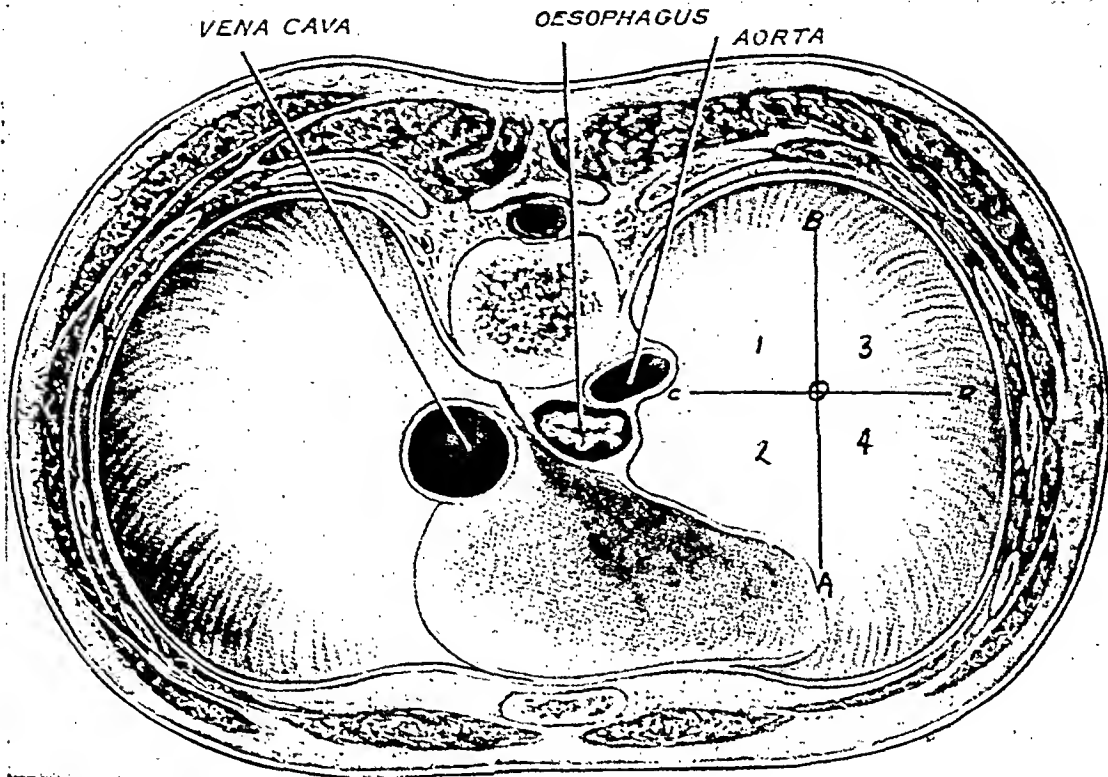
FIG. 1. Shows incisions of diaphragm.

uted to lack of eating because of distress in swallowing food. He had become progressively worse so that he was unable to carry out any of his duties as a janitor. The patient had noticed distress with a change of position or with the slightest pressure on the abdomen and occasionally was awakened at night because of severe dyspnea.

Physical examination revealed a well developed male with obvious signs of considerable weight loss. The blood pressure was 104/70, temperature 98.6°F., pulse 68; respiration 20. Detailed examination revealed bilateral inguinal hernia. The chest was tympanitic and the presence of gurgling bowel sounds and the absence of normal breath sounds in the left chest to about the level of the aortic arch were noted; extrasystole were present; otherwise the examination was essentially normal.

Laboratory examination showed a white blood cell count of 5,850. The hemoglobin was 107 per cent and the red cell count was 5,490,000. The color index was 94, the serum protein 7.0, albumin 4.9 and globulin 2.1 Gm. per 100 cc. The AG ratio was 2.3 to 1. The Wassermann and Kahn tests were negative. The urine was negative. An electrocardiograph revealed evidence of considerable myocardial damage.

Roentgenologic studies revealed an elevation

of the left dome of the diaphragm to about the level of the aortic arch. The heart was displaced to the right. A barium enema revealed the transverse colon at the level of the aortic arch and a large air bubble directly to the left of the displaced heart was noted. The diaphragm was elevated but of normal contour; the bowel and stomach were below it. (Fig. 3.)

The left chest and abdomen were prepared for operation and an ankle vein was used for intravenous injection. With the patient lying on his right side and with the left arm suspended from a rectangular frame, positive pressure ethylene-oxygen-ether anesthesia was administered through a snug fitting face mask. The left chest was opened through an incision over the eighth rib from the mammary line anteriorly to a point midway between the line of the angle of the scapula and the vertebral bodies, posteriorly. Excellent exposure was obtained.

The lung was retracted mesially and anteriorly, exposing the costovertebral gutter. The phrenic nerve was gently crushed to assure temporary paralysis of the diaphragm. The diaphragmatic pleura was incised anteroposteriorly at the mid-point of the distended dome, as shown in Figure 1 as the line A-B. This exposed the diaphragmatic musculature which was separated in the direction of its
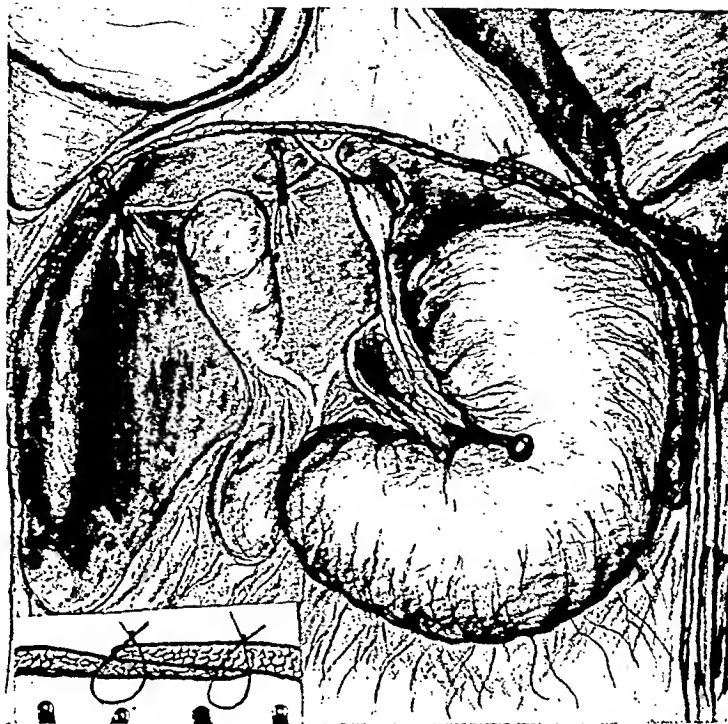
FIG. 2. Shows imbrication of leaves of incised diaphragm.

FIG. 3. Shows postoperative view of diaphragm.

fibers, thus exposing the diaphragmatic peritoneum. The peritoneum was then gently dissected from the lower surface of the diaphragm far enough anteroposteriorly and mediolaterally to allow the amount of imbrication of the dome necessary for the repair of eventration. The media leaf of the divided diaphragm was then incised lateromedially, as indicated by the line o-c of Figure 1, a distance far enough medially to allow the desired amount of imbrication necessary for correction of the anteroposterior arc of the diaphragm. The mediolateral leaf of the incised diaphragm, as indicated by line o-d of Figure 1, was treated in a similar manner. The incised leaves were then imbricated postero-anteriorly, as shown in Figure 2, with interrupted No. 00 chronic catgut. This corrected the excessive anteroposterior arc. The lateromedial arc was corrected by an imbrication of the mediolateral leaves as indicated in Figure 1 as 1-2 upon 3-4. Additional interrupted chronic catgut sutures were placed to secure the pleura over the lines of the incised diaphragm. A Pezzar catheter was inserted posterolaterally through the ninth interspace for pleural drainage. The wound was closed tightly.

Postoperatively, on November 14, 1946, the patient felt very well and was able to carry on hard manual labor without any distressing symptoms.

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HODGKIN'S DISEASE OF THE STOMACH*

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A REVIEW of the literature on Hodgkin's disease of the stomach reveals it to be a rare manifestation of the disease. Thomas Hodgkin[1] first described the disease as a clinical entity in 1832. It was not until 1913 that Schlagenhaufer[2] reported the first case of Hodgkin's disease in which the specific lesion was found in the gastrointestinal tract, namely, the stomach. According to Wallhauser,[3] isolated lymphogranuloma of the gastrointestinal tract is rather uncommon. Mead,[4] after reviewing 16,254 autopsies, reported only three cases in which there was isolated involvement of the gastrointestinal tract. Goldman,[5] analyzing 212 cases of Hodgkin's disease, found the stomach to be involved in only one case. Burger and Lehman[6] reviewed fifty-four cases of Hodgkin's disease and found only two stomach cases. Jackson and Parker,[7] reporting 174 cases found the stomach to be primarily involved in only three instances whereas the stomach was involved in only one of Goldman and Victor's[8] 319 cases of Hodgkin's disease.

The symptomatology is varied, indefinite and bizarre. In Jackson and Parker's[7] report on Hodgkin's disease, anorexia, abdominal pain, nausea, constipation, vomiting, diarrhea, dysphagia, melena and hematemesis are listed as symptoms of the disease in order of frequency. Our patient had epigastric pain, nausea, vomiting, one episode of hematemsis and persistent occult blood in the stools. Hayden and Apfelbach,[9] reviewing twenty-six cases and presenting three of their own, conclude that fever, diarrhea, abdominal pain, rapid cachexia, secondary anemia, leukopenia or a normal white blood count are the presenting signs and symptoms of the disease. They further state that the symptoms can be classified into three groups, namely, ulcerative enteritis and colitis, gastric carcinoma and intestinal obstruction. Our patient simulated the symptoms of gastric carcinoma. His white blood count was normal; he had abdominal pain but did not have intestinal obstruction. Madding[10] described six cases from the Mayo clinic. Two-thirds of his patients had involvement of the lesser curvature as did ours; fever was absent in our patient as it was in his. Bockus[11] states that an ulcer-like syndrome has been reported in several cases. He describes loss of weight, diarrhea, pruritus, jaundice and ascites as symptoms of this disease. He also states that skin pigmentation may be present. Our patient presented an ulcer syndrome over a period of two years. He had lost 19 pounds of weight during a four-year period. He had several episodes of epigastric pain, occurring a few hours after eating, which was relieved by the taking of food and antacids. He was treated as an ulcer patient over a period of two years with modified Sippy diet, antacids, antispasmodics and amino acids. He also had several small pigmented areas of the face and anterior chest wall which were thought to be secondary to an acne of long standing.

The roentgenologic findings are usually not of much help in establishing a definite diagnosis. Craver and Herman[12] state that the roentgenologic diagnosis is usually carcinoma or ulcer. In this case, our patient's symptoms were first diagnosed roentgenologically as an ulcer; later, an x-ray diagnosis of carcinoma of the stomach was made because of a persistent filling defect with ulceration. These findings are usually considered to be characteristic of carcinoma.

The diagnosis is difficult to make pre-

operatively. Sherman,[13] reviewing seventy-three cases of reports of gastrointestinal Hodgkin's disease, states that the disease usually simulates one of four main clinical entities: gastric carcinoma, gastric ulcer, enterocolitis or obstruction of the bowel. This is in agreement with Hayden and Apfelbach's[9] findings. He also mentions that the final diagnosis is established by necropsy or operation. The diagnosis of Hodgkin's disease of the stomach is usually made only on histologic section. In fact, Avent[14] states that the diagnosis of this condition is practically impossible to make before microscopic examination of the lesion is attempted. Feigenbaum[15] believes that there is no distinct clinical picture of abdominal Hodgkin's disease. It may resemble any intra-abdominal pathology, however, the general clinical features of Hodgkin's disease of the gastrointestinal tract simulates carcinoma which is the usual diagnosis made. In spite of the age of our patient, we thought we had been dealing with a malignancy which had developed at the ulcer site. The patient presented an ulcer syndrome over a period of two years with some relief from the usual ulcer therapy. The constant defect found on x-ray, together with the incomplete relief of symptoms on medical therapy, led us to believe that we were dealing with a malignancy which had developed at the ulcer site. The actual final diagnosis of Hodgkin's disease of the stomach was not made until the histologic sections had been examined.

The treatment recommended for Hodgkin's disease of the stomach is gastric resection.[11,12] Singer[16] reviewed six cases and reported one of his own. He states that isolated Hodgkin's disease of the stomach is an operable lesion and with removal of the diseased tissue the prognosis is good. Steindl[17] reported the first case of lymphogranuloma localized to the stomach in which an operation was successful. We performed a gastric resection on our patient and removed all of the diseased tissue.

CASE REPORT

E. N., a thirty-three year old, thin, white male, came to the Batavia Veterans Hospital on September 23, 1946, complaining of episodes of nausea, vomiting and a burning epigastric pain of two years' duration. He had weighed 131 pounds when he entered the army four years ago; his present weight was 112 pounds. During the period of his symptomatology he described his troubles as, "gas on the stomach" characterized by frequent belching and soft eructations. At various intervals he had mid-epigastric and left upper quadrant tenderness.

During the fall of 1944, he first noticed a burning sensation in the epigastrium, occurring two or three hours after eating, which was relieved by taking some food or drinking a glass of water or milk. The pain was not severe enough to awaken him at night. He reported to his unit surgeon who advised avoidance of fried foods, coffee and other such stimulants. Although the epigastric burning sensation persisted, it was less severe when following this regimen. In April, 1945, he vomited approximately 1 pint of bitter tasting material which was greenish in color. This was followed by episodes of vomiting four to five times daily. There was no blood in the vomitus.

He was then admitted to the station hospital where an x-ray was taken. According to the patient, the film was reported as negative. He returned to his unit and was symptom-free for a period of eighteen days. Vomiting began once again and so he was readmitted to the station hospital. Another x-ray was taken of his stomach and this time a diagnosis of gastric ulcer was made. His upper teeth were removed because they were infected and it was believed that this was contributing to his symptomatology.

He was then sent to a general hospital where he was treated with a bland diet, antacids and bed rest. During his stay in this hospital he experienced only a few episodes of burning epigastric pain and vomiting. When this occurred, he was relieved by taking 1 or 2 ounces of an aluminum hydroxide preparation.

In April, 1946, the patient observed that his burning epigastric pain was becoming more severe. It was accompanied by a siege of vomiting three to four times a day. This episode was of six days' duration. He had hematemesis on one occasion which was described by the

patient as a "mouthful of red blood along with other material."

At this time he was in Quebec and again hospitalized. He was again treated with antacids in milk, a bland diet and bed rest for nine days. When he was discharged, he was relatively asymptomatic. During May and June of 1945, while working as an attendant in a psychiatric hospital, the epigastric burning sensation returned with greater severity. It was accompanied by vomiting two to three times daily. He treated himself with antacids, taking them three to four times a day. He regulated the amounts according to the degree of pain and amount of vomiting. This continued until the time of his admission.

The past history was non-contributory. The patient had acne during his adolescence. A right inguinal herniorrhaphy was performed in 1940, with an uneventful recovery.

The physical examination revealed a thin, white, afebrile male, not acutely ill, weighing 112 pounds. The head and scalp was essentially negative. The pupils were round, regular, equal and reacted to light and accommodation. Extra ocular movements and the conjunctiva were normal. The ears and nose were externally negative. No upper teeth were found but the remaining lower teeth were in good condition; buccal and faucial structures and surfaces were normal. The thyroid was not palpable. There was no cervical adenopathy or venous engorgement in the neck. The thorax was normal in size and shape. The lungs were normal to palpation, percussion and auscultation. The blood pressure was 105/50. The heart borders were within normal limits. The pulse was regular and of good quality; no arrhythmia, murmurs or friction rub were present. The herniorrhaphy scar in the right inguinal region was well healed and not tender; there was a small area of tenderness in the left upper quadrant but no masses were palpable in the abdomen. The liver and spleen could not be felt. Examination of the external inguinal rings revealed no hernia. The genitourinary system was externally negative and the rectal examination was also negative. There were no palpable lymph glands. There was no orthopedic imbalance, asymmetry or limitation of motion of the musculoskeletal system. The patient's reflexes and reactions were normal.

The laboratory findings were as follows: The Wassermann and Kahn tests were negative. The hemoglobin ranged from 76 to 92 per cent; the red blood count varied from 4,040,000 to 4,800,000 and the white blood count varied from 7,600 to 9,800. The differential leukocyte count on several occasions showed 73 to 79 per cent polymorphonuclears, 18 to 24 lymphocytes, 3 monocytes and no eosinophiles. The blood proteins and blood chemistry were within normal limits. The coagulation time was four and one-half minutes and the bleeding time two minutes. The hematocrit was 46 per cent and the vitamin C level 2 mg. per cent. The sedimentation rate was 24 mm. per hour. Urinalyses were normal; gastric analysis (Ewald test meal) on September 28, 1946, showed 50 degrees total acid, 40 free and 6 combined. On October 22, 1946, the patient had 30 degrees total acid, 20 free and 6 combined. Repeated examination of the stools showed occult blood but no ova or parasites.

X-ray findings were as follows: September, 27, 1946, during the gastrointestinal series the stomach showed a slight projection on the lesser curvature which was not locally tender but visible on all films in the posterior surface. The six-hour status showed a small gastric residue. October 11, 1946, the stomach showed a small filling defect associated with a shallow ulceration on the lesser curvature at the angle. There was tenderness in this region. A number of films made with and without compression showed a persistent rigidity and straightening in this region. Comparison with previous films showed that not only was there a lack of healing in the gastric ulcer then described but also as a result of compression studies a filling defect was evident. The appearance suggested carcinoma of the stomach. A chest plate was taken on September 27, 1946 and was reported as negative. This was repeated on October 23, 1946 and was also reported as negative. No abnormality in the lungs, ribs and no mediastinal lymphadenopathy was noted. Gastroscopy was performed on October 17, 1946 and substantiated the x-ray diagnosis of carcinoma of the stomach.

Abdominal exploration was recommended with gastric resection if the lesion was resectable. The patient was prepared preoperatively with blood transfusions, intravenous amigen, glucose, saline, vitamins and a Levine tube with Wagensteen suction. On October 24, 1946, an exploratory laparotomy was performed which revealed a large mass on the lesser

curvature of the stomach just above the angle. There were a few palpable lymph nodes present in the gastrohepatic and gastrocolic ligaments. There was no involvement of the mesenteric nodes. The liver, spleen and small and large intestine were all normal. The mass was found to be resectable. A subtotal gastric resection was performed with gastrojejunostomy (anterior Polya type).

The section of stomach removed showed a large superficial ulcer of the lesser curvature with thickening of the wall around it. The thickening of the wall was due to a soft, yellowish-white deposit beneath the mucosa which caused the latter to be flattened out. This was an ulcerated polypoid growth measuring 6 cm. in diameter and infiltrating the stomach wall widely about it. The shallow ragged ulcer crater measured 3.5 cm. There were five lymph nodes along the lesser curvature, varying in size from a pea to a lima bean, and also three lima bean sized nodes along the greater curvature of the stomach. On section, these were yellowish-white in color and firm.

Microscopic sections through the gastric tumor revealed a fairly typical picture of Hodgkin's granuloma. The granulomatous tissue diffusely infiltrated the surrounding submucosa, infiltrated between the underlying muscle bundles and muscle layers into the mucosa causing atrophy of the gland tubules. Section from the floor of the ulcer showed superficial peptic necrosis, acute inflammatory infiltration and edema. The serosa showed unusually marked edema throughout with occasional scattered focal collections of lymphocytes and plasma cells. The granulomatous areas revealed the usual reticulum cell hyperplasia together with a few Sternberg-Reed giant cells, many lymphocytes, plasma cells, a few polymorphonuclear leukocytes and eosinophiles. Lymph nodes also revealed the same pathologic condition as in the granulomatous areas.

The postoperative course was uneventful. The patient was given blood, glucose, amigen and saline intravenously. Small feedings of 1 ounce of water were started on the second postoperative day and gradually worked up to a modified Sippy diet. The wound healed per primum and the patient was out of bed on the fifth day. After recovery the patient was transferred to the Bronx Veterans Hospital for further treatment in their tumor clinic where he will have deep x-ray therapy and administration of nitrogen mustards.[18]

SUMMARY

We have presented a case of Hodgkin's disease of the stomach; this is a rare entity. Our patient presented an ulcer syndrome with episodes of abdominal pain, nausea, vomiting and a weight loss of 19 pounds during a four-year period. The history is similar to that of other cases reported. The physical examination was essentially negative except for left upper quadrant tenderness. No lymph nodes were palpable in the cervical, axillary or inguinal regions. The positive laboratory findings were an elevated sedimentation rate of 24 mm. per hour and persistent occult blood in the stools. Roentgenograms showed a constant filling defect with ulceration of the lesser curvature of the stomach. X-ray diagnosis of carcinoma of the stomach was confirmed by gastroscopy. The patient was treated by a subtotal gastric resection and gastrojejunostomy. All of the diseased tissue was removed. The final diagnosis was made by histologic examination. We believe the prognosis in this case to be good. Deep x-ray and nitrogen mustard therapy has been advised.

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A. S. LENTINO studied five cases of myasthenia gravis and the thymus and found that biopsy showed a malignant tumor (thymoepithelioma) and a benign tumor (adenoma) in two patients and in the remaining three patients there was hyperplasia of the thymus gland. The author, therefore, concludes that a distinct relationship exists between an altered thymus and this disease. (RICHARD A. LEONARDO, M.D.)

ECTOPIC BONE DEPOSITS*

A PARAPLEGIC COMPLICATION

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LITERATURE dealing with injuries of the spinal cord, the subsequent paralysis and complications is growing rapidly. Emergency handling and transportation, the diagnostic phase of neurologic and orthopedic appraisal, whether or not and when to operate, problems of urinary management, occurrence and prevention of decubitus ulcers, nutritional problems, importance of skilled, sympathetic and continuous nursing—all these matters have been dwelt upon thoroughly in other places and will not be repeated here. Accent has been placed upon each in turn, and suffice it to say, each and all must be successfully managed.

Satisfactory rehabilitation means salvaging as much of a tolerable and productive future for the patient as the severity of his affliction will allow. It means he must have successfully escaped the hazards of urinary sepsis and decubitus disease and has won out to at least a wheelchair existence. There is a paraplegic complication mentioned intermittently in recent literature which merits much more emphasis. If at least a wheelchair existence is the goal toward which all the intensive and tedious medical and surgical measures are directed, this paraplegic complication can and on occasion does obstruct that objective.

In an issue of the U. S. Army Bulletin a preliminary report[1] appeared. In this report a study was made of sixty-two cases of paraplegia resulting from disease or injury of the spinal cord or cauda equina. Twenty-three of these patients showed ectopic deposits of trabeculated bone within the muscles or fascial planes, principally over the hip and knee joints. Those patients showing degrees of recovery in

function seemed to escape this additional complication for only three developed osseous deposits. Of thirty-five unimproved cases, twenty showed ossifications. An analysis of dermatome levels in the patients with ossification showed no predilection for any particular level. Nor did there appear to be, in comparison with the unaffected group, any "significant difference with regard to sites or severity of injuries, time of operative treatment, presence or absence of decubitus ulcers, urinary tract infection or associated injuries or infections, or blood calcium and total protein levels." Ossifications were said to be recognized as early as forty days following cord injury.

The following clinical case is cited to call attention to the important fact that paraplegics are not out of the woods and into the clearing of manageable convalescence and rehabilitation if the complication of ectopic bone deposits is not anticipated and an effort made to prevent their development.

CASE REPORT

In June, 1945, a patient was admitted to the Naval Hospital at which one of us (M.B.R.) was stationed. He was a twenty-two year old male, navy veteran with paralysis of both lower extremities, a decubitus ulcer over his sacrum and inability to sit normally or comfortably for any period of time in a wheelchair. While in boot-camp training he had sustained an acute onset of illness on February 14, 1943, with rapidly progressing paralysis of both lower extremities. A diagnosis of extradural inflammatory process was made and a laminectomy of the third, fourth, fifth and sixth dorsal vertebrae was performed in March, 1943, which verified the diagnosis. Convalescence

FIG. 1. G. D. D., age twenty-two years; antero-posterior view. Massive bridges of coarsely trabe-culated but otherwise normal appearing bone extend from the anterior portions of both ilia and the lower rims of both acetabuli to the anterior portions of the head, neck and upper fourths of both femora. The structure of the hip joints and femora are readily seen through the abnormal bone masses and present a normal appearance except for advanced atrophy. The hip joint spaces are not narrowed. On the left side some of the bone mass appears fractured medial to the femoral neck.

had included periods at two veteran's hospitals until July, 1944, at which time he was discharged to his home.

The paralysis of both lower extremities, present before the operation and persisting thereafter, showed little alteration as time went on. A finding of note in his physical examination was the presence of solid, hard masses over the anterior aspects of both hip joints. The right hip joint was solidly ankylosed in about 30 degrees of flexion while on the left there was an additional 10 degrees due to as much range of motion. The patient dated the onset of his acute illness as of February 14, 1943. From a transcript of his history the statement was noted that on March 10, 1943, he noticed a swelling for the first time developing over his right hip which became progressively larger. This would date the appearance of the ectopic bone deposits as less than four weeks after the acute onset of the illness. During the course of his convalescence he was fitted with double, upright braces. With these and the aid of crutches he was able to walk tripod fashion across the room and for a distance along the corridor. Fatigue of his upper extremities, of

course, made this a self-limiting activity. He was able to move his bowels by means of abdominal pressure and to empty his bladder by means of manual compression.

Roentgenographic studies of his dorsal vertebrae disclosed the surgical absence of most of the laminae and spines of the third, fourth, fifth and sixth dorsal segments. There was no abnormal soft tissue calcification or ossification. The anteroposterior (stereoscopic), (Fig. 1) and lateral views of the lumbar spine and pelvis showed massive bridges of coarsely trabeculated but otherwise normal appearing bone extending from the anterior portions of both ilia and the lower rims of both acetabuli to the anterior portions of the heads, necks and upper fourths of both femora. The structure of the hip joints and femora could be readily seen through the abnormal bone masses and presented a normal appearance, except for advanced atrophy. The hip joint spaces were not narrowed. On the left side some of the bone mass appeared fractured in the region medial to the femoral neck.

The patient spent much of his time lying in bed between his ambulatory excursions rather than in a wheel-chair. This preference could not be discouraged nor, on the other hand, could he be prevailed upon to sit for any prolonged period in a wheel-chair. Because of the extension deformity of his hips such sitting was only possible by using the full flexibility of his lumbar spine. He sat on his sacrum rather than on his buttocks and hence the persistently recurring decubitus ulcer. If the complication of ectopic bone about the hips had not intervened, this patient would certainly have been an example of satisfactory rehabilitation.

As a result of such and similar observations, the authors thereafter and in private practise have been making special efforts to forestall the development of this complication in the management of paraplegics. It is their belief that the setting of the ossification pattern, if it develops, will do so in the early stages of recumbency; that is, in the immediate interval of bed rest following the onset of paraplegia and until such time as the patient is able to be sat up and later stood up for training in tripod walking. As was noted, this may be within the first four to six weeks of re-
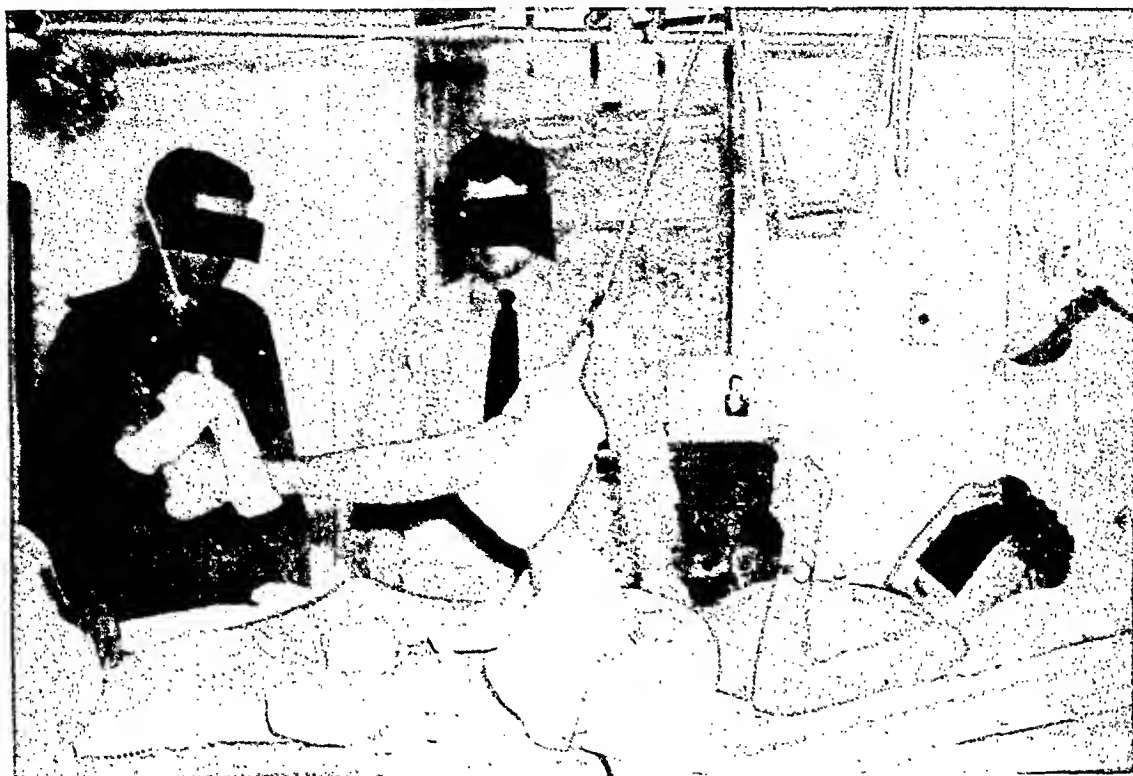
Fig. 2. Narrow slings of softly padded moleskin flannel are made to fit about the heels and around the knees of the patient. These are attached to cords suspended from pulleys and disposed in such a manner that the patient himself by exerting traction can lift each extremity until the ankle, knee and hip joints flex to a complete 90 degrees. Soft pillows are placed under the thighs and legs so overlapping as to allow 5 to 10 degrees of flexion of the knees and hip joints. Rolled pillows are placed between the feet and the foot of the bed to maintain the former at right angle position to the legs. The pillows beneath the legs are sufficient thickness to prevent the heels from lying directly upon the bed. To prevent continuous internal rotation and adduction contractures of the lower extremities, a small well padded block of proper dimensions is placed between the knees. The head of the fracture bed is raised slightly to relax the abdominal muscles.

cumbency. Mobilization as early as possible would seem to be the key-note.

During the initial period of recumbency, it has been the practice to begin physical conditioning of the patient from the very beginning. Much of this effort has been directed toward the development of the upper extremity, shoulder girdle and trunk muscles in anticipation of the eventual management of crutches in tripod ambulation. Because of the possibility of ectopic bone deposits ankylosing the hip joints and perhaps less frequently but just as definitely the knee joints, the powerless lower extremities should just as assiduously be carried through a complete range of motions in a passive manner. This is accomplished by a simple and time-honored device. Narrow slings of softly padded moleskin flannel are made to fit about the heels and around the knees of the pa-

tient. These are attached to cords suspended from pulleys and disposed in such manner that the patient himself by exerting traction can lift each extremity until the ankle, knee and hip joints flex to a complete 90 degrees. (Fig. 2.) This is his task in the program of his own rehabilitation; he believes he is helping to accomplish his own come-back; and because he enjoys doing it, it is good for his morale. Nor does he have to depend upon others to get this particular job done.

The patient is placed on a two-segment air mattress in a fracture bed. Soft pillows are placed under the thighs and legs so overlapping as to allow some 5 to 10 degrees of flexion of the knees and hip joints. Rolled pillows are placed between the feet and the foot of the bed to maintain the former at right angle position to the legs. The pillows beneath the legs are of suffi-

FIG. 3. J. E. S., age thirty years; anteroposterior view. There is a large amount of ectopic bone lying in the soft tissue anterior to the inferior portion of the ilium, the hip joint and along the medial aspect of the right femur, ending just below the lesser trochanter; traumatic transection of the cord sustained five months previously.

cient thickness to prevent the heels from lying directly upon the bed. To ward off continuous internal rotation and adduction contractures (both deformities being encouraged by the intermittent spastic seizures), a small well padded block of proper dimensions is placed between the knees. The head of the fracture bed is raised slightly to relax the abdominal muscles. For a period in the morning and again in the afternoon, the patient is transferred from the fracture bed and laid face downward upon a cart. The prone position on the cart with feet resting downward over the end, provides complete extension of the knee and hip joints, also an ample period of time for skin care. The cart can then be moved to the sun porch or such other place as will provide a change of scene.

As soon as is feasible, this routine of resting face downward on a cart is eliminated by getting the patient up in a wheelchair. He is then able to get about under his own power to visit about the ward and to go outside for varying periods when the weather allows. During the interval of recumbency, his lower extremities are measured for braces and, after a reasonable period in a wheel-chair, he is next fitted with his braces and, first in a walker and eventually with crutches, he is taught to ambulate tripod fashion. While no prolonged periods of walking are possible, the general stimulation of the upright position and weight-bearing both to his general health and to his morale is extremely worth while. It is yet another means of defeating the untoward influences of recumbency and, therefore, of the tendency for a continued decalcification of his bones.

General immobilization for a prolonged period depletes the calcium store of the whole skeleton, as has been observed in x-rays, and by the abnormally increased amounts of calcium in the blood stream and urine. The byproduct of this hypercalcemia and hypercalcinuria is the development of calculi in the renal pelvis and ureters. Whether there is a further reflection of this pathologic process in the formation of ectopic bone remains to be demonstrated. Some would add a neurogenic factor. But we have yet to encounter such a complication as ectopic bone about the hip joints in paraplegic cases resulting from anterior poliomyelitis. In the light of our present knowledge, it is therefore reasonable to attack the problem and forestall the appearance of this complication by eliminating the factors which tend to mobilize skeletal calcium, enforced recumbency and generalized immobilization.

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PRIMARY TORSION OF THE OMENTUM

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ONE of the conditions seldom considered in the differential diagnosis of the acute condition of the abdomen, and partly for that reason seldom diagnosed preoperatively, is primary torsion of the omentum. If the publication of individual case reports serves no other purpose, it at least acts as a reminder to the reader that, although not a common clinical phenomenon, primary omental torsion merits some consideration in surgical diagnosis, especially when the history and clinical picture do not point conclusively to any of the more common abdominal lesions.

Omental torsion may be roughly defined as a twisting or rotation of either the whole or a part of the omentum upon itself, resulting in the formation of a strictured neck distal to which the circulation is obstructed by the constriction. For practical purposes two types may be considered: an uncommon, primary, cryptogenic or idiopathic type in which there is no apparent cause for the torsion, and a second, more frequently encountered type resultant from or associated with a pathologic condition within the abdomen or in some external hernial sac. In 1928, McWhorter[1] compiled from the literature twenty-eight cases of the primary variety. This series was increased to thirty-nine in 1930 by D'Errico[2] and to fifty-four by Andrews[3] in 1938, to which Anton[4] has added three additional cases.

Various concepts have been advanced to explain the mechanism of primary torsion but only three merit consideration:

1. In view of the history of trauma noted in many patients some observers have contributed the view that importance be attached to external violence, severe exercise or strenuous body movements as the motivating forces initiating the rotation and twisting of the omentum.

2. Inasmuch as the omentum is constantly being rolled and twisted about as the result of intestinal peristalsis, especially when it is interposed between two coils of gut, gut and other abdominal viscera or the abdominal wall, it is highly conceivable that torsion might be induced by this factor. This is the most plausible of all three theories.

3. An ingenious but highly dubious hemodynamic theory was put forth by Payr[5] who maintained that inasmuch as the omental veins are larger, longer and more tortuous than the arteries and more easily compressed, any kinking of the omentum will result in some impediment to the venous circulation resulting in venous engorgement. This engorgement necessitates an increased pressure in the arteries which become tense, and the engorged veins in an effort to accommodate themselves wind around the artery, thus causing the whole omental mass to undergo twisting.

Whatever may be the mechanism involved in torsion, the omentum twists in a clock-wise direction, the number of twists being variable. The circulation is interfered with, edema and extravasation of blood occur into the mass and there is an effusion of serosanguineous fluid into the abdominal cavity. Grossly, the normally yellowish omentum becomes reddish with engorgement, purplish with extravasation and finally black with gangrene. The gangrenous omentum then sets up an inflammatory reaction in the surrounding tissues which develops into an aseptic peritonitis, or else organisms from the adjacent bowel may penetrate the necrotic

mass to produce an abscess. Rarely, the mass may undergo atrophy and become partially or entirely absorbed, or it may become auto-amputated and attach itself to some neighboring structure.

The condition is rarely diagnosed pre-operatively because it is seldom thought of in the differential diagnosis of the acute abdomen, and with the exception of the recognition of a mobile diffuse tumor mass early in the disease, there is nothing in the clinical picture which might distinguish it from the many other causes of the acute condition of the abdomen. Such signs and symptoms as nausea, vomiting, abdominal tenderness, fever, pulse acceleration and leukocytosis which do occur with primary torsion of the omentum are the early signs of any acute intra-abdominal lesion. For that reason most of the cases are diagnosed as appendicitis, gallbladder or gastroduodenal disease, or twisted ovarian cyst.

The treatment of the condition is simple. If on opening the abdomen one finds serosanguineous fluid, the search for omental torsion is mandatory.[3] Excision of the mass proximal to the point of rotation, with serial ligation of the omentum, is the indicated operative procedure.

CASE REPORT

K. R., a twenty-two year old soldier, was admitted to the Surgical Service of the hospital November 13, 1945 with a history of becoming ill on the afternoon of November 11th. This illness consisted of a sudden attack of what the patient called indigestion. There was no belching, no nausea and no vomiting, but only a feeling of epigastric distress and fullness. This feeling remained localized in the upper abdomen and the umbilical region. The pain remained all day as a dull ache in the epigastrium. He slept well that night but awoke the next morning with the same pain which lasted all day without remission and was not relieved by a Bromo-Seltzer. However, the pain was not intense enough to interfere with the patient's eating. His bowel habits remained normal. He slept well that night but on the morning of November 13th he awoke with more intense pain than on the previous two days. He reported to his unit dispensary from which he was sent to the hospital with a diagnosis of appendicitis. At no time had there been any nausea or vomiting. When the patient was examined on admission, his temperature was 100.6°F., pulse 124 and respirations 24. His white blood cell count was 10,000; polymorphonuclears, 70 per cent; lymphocytes, 28 per cent and eosinophiles, 2 per cent. There was some generalized abdominal tenderness, no masses and no spasm. Tenderness was most marked in the right lower quadrant. The ward surgeon did not think the patient's condition at this time warranted surgical intervention. The next morning his temperature was 99.8°F., pulse 82 and respirations 20. The white blood cell count was 7,600; polymorphonuclears, 67 per cent; lymphocytes, 31 per cent and basophiles, 2 per cent. Examination at this time showed increased tenderness in the right lower quadrant with some muscle spasm together with generalized abdominal tenderness. No masses were palpable and rectal examination was negative. Operation was decided upon, the tentative diagnosis being acute appendicitis. The abdomen was opened through a McBurney incision. On opening the peritoneum there was an escape of serosanguineous fluid. There were coils of redundant sigmoid in the right iliac fossa and the appendix when brought into view was normal. The incision was enlarged to permit the entrance of a hand for exploration of the abdomen. The rectum, sigmoid, descending colon, splenic flexure and transverse colon were normal to palpation. In the right upper quadrant a firm mass about the size and shape of a baseball was palpated. This mass was easily brought down into the wound and found to be a purplish, globular mass 8 cm. in diameter, the result of a 360 degree clock-wise rotation of the distal portion of the omentum. The mass was excised by serial ligation of the omentum proximal to the point of constriction. Appendectomy was done and the abdomen closed without drainage. The patient made an uneventful recovery.

Comment. The absence of nausea or vomiting should have made one suspicious of the diagnosis of acute appendicitis.

SUMMARY

1. A case of primary torsion of the omentum is presented.

2. The condition is briefly discussed as to frequency, mechanism, diagnosis and treatment.

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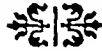
The "overprolonged" use of the Miller-Abbott tube in intestinal obstruction may cause intestinal perforation, fatal or otherwise. Berger and Achs report a recent patient that recovered but surgeons and especially the hospital interns and residents, must surely realize that passage of this hard rubber tube may be dangerous, especially if the vitality of the bowel is questionable. They must, therefore, be careful in its use. Properly used, in the vast majority of cases, it is often a life-saving procedure. It is well, however, to keep its occasional dangers in mind. (Richard A. Leonardo, M.D.)

HIGH INTESTINAL FISTULA AND ITS TREATMENT BY THE USE OF A PAULS TUBE*

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INTESTINAL fistula is an irritating and troublesome complication of many surgical procedures; it is usually dangerous to life only when it occurs in the proximal part of the small bowel. Fistulas of the jejunum and duodenum are so very disabling that in a short space of time the patient is reduced by loss of fluids, electrolytes, chlorides and nourishment to a state of complete collapse. There ensues a rise in blood nitrogen and a corresponding fall in blood sodium and chloride ions. Such patients are quite unable to stand any operative interference due to the extreme state of emaciation and debility; their general condition is aggravated by a severe excoriating erosion of the surrounding skin. Urgent treatment is imperative. The patient must receive a definite amount of nourishment, otherwise starvation and dehydration follow very rapidly and death ensues. The problem is extremely acute and will tax the ingenuity of the surgeon to the highest degree.

Some means must be found whereby the fistula may be closed, but in such a way that the lumen of the bowel remains unimpeded. Reybard used a wooden button in the lumen of the gut tied to a crossbar on the abdominal wall by a suture passing through the fistula. A button of leather was recommended by Kleybolte. Dowd described a double metallic button which has also been successfully used by Mayo-Kappis in 1911. A T tube was used with the straight part of the tube providing a channel between the upper and lower parts of the bowel with the right-angled part protruding through the fistula blocking it and providing drainage for overflow.

These methods are all good in certain limited cases. It often happens, however, that the fistula is situated in a part of the bowel that makes an acute angulation; therefore, no button is able to maintain its position and will be displaced almost at once. The disadvantage of the T tube is that there is a constant flow of intestinal contents around it. This produces a fatal outcome, especially if the fistula is a large one. We are thus driven to the conclusion that these methods have a very limited field and are useful in those types of fistula only in which the opening occurs as a defect in a horizontal segment of bowel.

There may be, however, a complete severance of continuity of the small bowel or the opening may be a very large one. In these cases inadequate treatment is rapidly fatal because the patient is deprived of all nourishment. To meet such an emergency as the above, Macnaughton has devised an apparatus which was successfully used by him and appears to me to be an excellent device. Rubber catheters are inserted, one into the afferent and the other into efferent segments of the bowel. The intestinal contents by means of continuous suction are aspirated into a container from which they are allowed by force of gravity to flow into the efferent catheter and so into the distal bowel. This device requires a certain amount of ingenuity to construct, but undoubtedly appears to be the best method as yet described for overcoming this very acute surgical complication.

Recently a patient developed a very high jejunal fistula, a fistula that was so large that no intestinal content could pass beyond the opening in the bowel and a state of extreme urgency rapidly developed; the

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patient became almost pulseless and it was obvious that any further delay would be fatal. Rubber buttons and tubes were inserted; but because of the angulation of the bowel and because of the great size of the fistula, they came out at once. It occurred to me that a Pauls tube might temporarily close the opening and at the same time divert the jejunal contents into the distal bowel; further than that Pauls tubes are obtainable in three sizes and so one of these tubes would most likely fit the bowel snugly. In addition, since they are constructed in the form of a right angle, they would be eminently suitable inasmuch as the point of opening of the fistula coincided with the apex of the angulated bowel. The smallest of the Pauls tubes was found to be of the correct size, so a stout piece of silk was tied around the middle of the tube and left long; the Pauls tube was fairly easily manipulated into the bowel through the fistulous opening and maintained in position by anchoring to a wooden tongue depressor on the abdominal wall. The bowel leakage was reduced to a minimum and the fluid contents of the jejunum could be seen passing along the Pauls tube into the distal segment of the bowel. (Fig. 1.)

The patient at once reacted in a most remarkable manner; whereas before he had appeared to be almost moribund and in a semicomatose state, he became a different being altogether within a few hours. His pulse became strong and slow and he was able and willing to drink freely; his bed which had been constantly soaked with intestinal fluids remained dry and only the dressings became somewhat soiled. Some 90 per cent of the jejunal contents were able to pass into the distal bowel. Every few days the Pauls tube would become clogged with solid matter, and it was found to be an easy procedure to remove and clean the glass tube and to reinsert it. In the course of a few weeks, the patient regained his health and strength to such an extent that to all outward appearances he was a perfectly normal man.
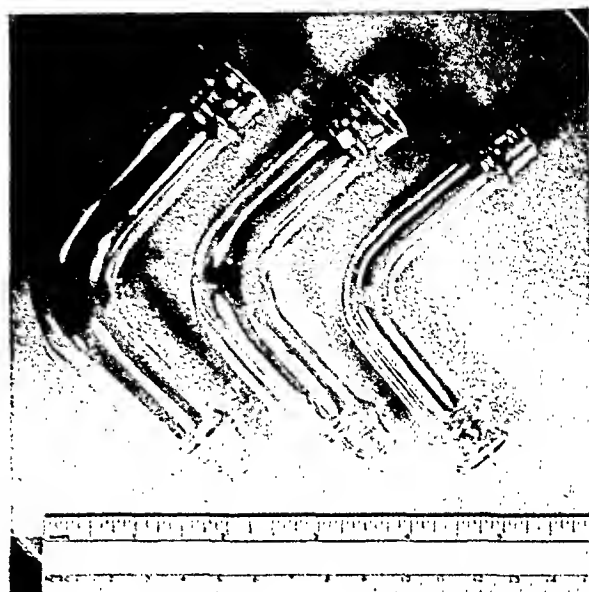

Fig. 1. Illustration showing the three sizes of the Pauls tube.

At this stage an operation was performed with the Pauls tube still in place; the two ends of the bowel were freed up and mobilized, the peritoneal cavity was opened and an end-to-end anastomosis carried out.

CASE REPORT

A man, sixty-five years of age, was admitted to the hospital complaining of nausea, heartburn, and a feeling of weight in the epigastrium of six months' duration. His condition steadily deteriorated and was accentuated by loss of appetite and loss of weight. He had suffered for many years with a severe form of asthma. His family history indicated that the father had been a victim of asthma. X-rays showed a carcinoma near the pyloric end of the stomach; the tumor was of moderate size.

Physical examination showed an elderly man somewhat debilitated, breathing asthmatically. The color of the skin and of the mucous membrane was normal. The tongue was moistened but not coated. Bronchial râles on both sides were heard. The abdomen was soft with tenderness in the epigastrium. The liver and spleen were not enlarged. There was no edema of the extremities. Urine examination was negative. Examination of blood was as follows: Blood urea 28 mg. per cent; sugar 101 mg. per cent; Kahn test negative; total protein 6.95, albumin 4.03, globulin 2.92. On March 3, 1946, subtotal gastrectomy was carried out under continuous spinal anesthesia; following the operation he coughed a great deal and contracted a severe bronchitis. On March 11th, the stitches were

removed, and some hours later he coughed violently and an evisceration occurred.

Operation was at once performed and the abdomen was closed with through-and-through stitches of heavy silk; a large penrose drain was placed between the silk stitches and the bowel in order to prevent pressure of the silk producing a fistula. On March 21st, a fistula developed with the outpouring of a vast quantity of jejunal content. Some days later the surrounding skin became macerated so a catheter was inserted and continuous suction instituted.

The patient's health and strength rapidly declined. He lost weight, became emaciated and dehydrated in spite of continuous intravenous saline and glucose infusions and many blood transfusions.

On April 7th, operation was performed. The fistula was closed and covered with omentum. The closure was only temporary and his general condition still further deteriorated. The patient became almost moribund.

On April 21st, a Pauls tube was inserted thereby connecting the afferent and efferent ends of the jejunum. It was kept in position by a silk thread attached to a wooden applicator. The jejunal contents at once began to pass along the Pauls tube and, as a result, the general condition of the patient began to improve although the artificial junction gave rise to some leakage; yet, he rapidly picked up strength to such a degree that on May 14th an entero-entero anastomosis was performed, and the patient left the hospital on May 29, 1946, in excellent condition.

CONCLUSION

Fistulas of high intestinal origin are a source of great danger. Immediate radical treatment cannot be carried out because of the condition of extreme emaciation that almost immediately ensues. A method is described wherein the patient obtains sufficient nourishment that will permit him to gather strength to undergo a major operative procedure.

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Editorial

WOUND HEALING AND NOSTRUMS

ACCORDING to a standard dictionary, a nostrum is "a medicine recommended by its preparer, especially a quack medicine." Nostrums can be divided into two classes, those which are recommended by their preparers directly to the consuming lay public and those which are recommended to a middle man, the physician. In the first classification may be found such boons to mankind as reducing salts, bust developers, alcohol repellents to be put in coffee and pills to cure self-diagnosed kidney trouble. The outstanding example of a nostrum in the second group is the proprietary preparation which is alleged to affect favorably the healing of wounds.

A little reflection will show why it is that the nostrum preparer has found such green pastures in the field of wound healing. He is continually favored by the strong biologic tendency of the body to heal itself. In particular, small incised wounds and minor burns are quite apt to heal regardless of what is done for them.

Let us consider the steps in the development of a market for a new burn ointment. It is only necessary to select a jelly or grease base which is not definitely harmful and which does not produce pain when applied locally. To this is added minute quantities of the factor for which therapeutic properties are claimed, after which it is neatly packaged in tubes or jars. Before the product can be sold permission must be obtained from the agency administering the U.S. Pure Food and Drug Law. For this it is necessary to secure a number of case reports from physicians who have tried the ointment. Therefore, a few hundred pounds of the product are shipped to doctors who conduct what is later referred to in the advertising brochures as "painstaking clinical research." The ointment is applied to some second degree burns which heal in a week or two, and the observers are likely to be of the impression that the progress of the lesions was somewhat better than had been observed with other methods of treatment. The preparer gathers up these case reports, presents them to the government agency, and sooner or later gets the nod that the marketing may begin. The commercial phase of the process will be facilitated by the publication of the case reports in an article entitled "A New Treatment for Burns: A Clinical Study of 49 Cases." Reprints of the article can be sent along with the more colorful promotion material.

The sequence of events just described must have been repeated many times because a great number of burn remedies are on the market. Several years ago a study was made of the methods of treatment of minor burns in three large industrial con-

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cerns. It was found that in the care of 5,609 burns, 84 different substances were used. Fifty-seven of these carried the brand names of their preparers. Statistics on the days required for healing showed that it made no difference what was put on these minor burns; if there was an advantage, it was with the simple non-medicated preparations.

The basic defect in the so-called clinical research on substances used for the treatment of wounds is that controls are usually lacking. It may be assumed that in the hypothetical paper mentioned above all the forty-nine patients were treated in the same way, with the new ointment. The proper approach would have been to treat the alternate patients with a presumably inert substance. In all probability the results would not have been statistically significant but if several other investigators were to publish similar controlled studies, the aggregate of all of the reports might provide a basis for real evaluation.

There are several more or less standard types of wound healing experiments which are available to make critical tests of substances for which claims of stimulation of healing are made. Small, symmetrical cutaneous wounds can be made in the guinea pig; the substance to be investigated is placed on one wound and nothing is placed on the other. No agent has been found which will consistently cause the treated wound to heal more rapidly than the control. In the human a convenient test wound is the dermatone donor site where wounds of the same size and depth may be made on symmetrical areas of the body. Again, there is no report of a substance which will accelerate the healing of this partial thickness wound.

A few years ago a popular news magazine reported a "New Burn Cure." The preparer, appraising his nostrum, had stated for the record that the research work in connection with the new treatment would "go down in history as second to none other, including the work of Pasteur." Although controlled wound healing experiments by individuals other than the preparer have shown that the ointment has no action not possessed by plain petrolatum, the advertising copy still mentions "Acceleration of rate of healing and epithelization".

CONRAD R. LAM, M.D.

Original Articles

TREATMENT OF FRACTURES OF LONG BONES BY OPEN REDUCTION AND SCREW FIXATION*

A REPORT OF FORTY-TWO CASES

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IT is the purpose of this paper to report on the successful treatment of forty-two fractures of long bones by operative reduction and screw fixation. In the majority of these fractures the ends of the fragments were without sufficient serrations or the displacing muscle forces were too strong to permit obtaining satisfactory reduction and control of the fragments by the routine manipulative and plaster fixation method. Because of the disadvantages and inadequacies of the popular traction procedures, this form of treatment was not considered, and in a few cases that came late to the author's attention traction had already proved to be unsuccessful. Screws were preferred to plates for fixation material because of the ease of execution of screw fixation and the adaptability of screws to different types of fractures as well as to fractures in the region of joints.

DISADVANTAGES OF TRACTION METHODS

Among the requisites of balanced suspension traction is constant supervision in order to ensure the maintenance of the fragments in correct position. This is a drawback to the method since it is often difficult to provide for such supervision, especially when fractures are treated in distant hospitals and can be inspected only periodically, as was true of many cases in this series. Furthermore, this balanced traction method calls for prolonged hospitalization and an extended period of discomfort and inactivity, particularly in fractures of the lower extremity.

Another limitation of both weight traction and double pin traction methods, which is being recognized more and more, is distraction of the fractured surfaces. This is a factor most inimical to the production of callus. Even a minimal amount of separation between the main fragments is sufficient to cause a delay in healing. The distraction is usually the result of over-pulling the fragments in an attempt to obtain close apposition and alignment. When the fracture is of the spiral type and distraction is present, the line of thrust is shunted out on both sides instead of up the center of the bone along the normal line of thrust, thereby retarding the formation of callus.

With the use of traction an angulation of the fragments is likely to occur which, in turn, causes an uneven distribution of callus and delayed union. Slow healing due to angulation is seen in fractures of the femur with bowing in which callus forms in the concavity of the bow but not on the convex side.

ADVANTAGES OF SCREW FIXATION

The open method which provides effective rigidity offers a much safer course of treatment than traction, when the patient cannot be under the operator's supervision following the reduction of the fracture. Only a minimal period of hospitalization is

required following screw fixation. After the initial swelling has subsided in a few days the patient has no complaint. In the average case of a fracture of the femur that has been transfixed the patient is out of bed within a few days and may be discharged from the hospital within two weeks. Since the average period of hospitalization for a fractured femur treated by traction is eleven weeks, the economic loss is cut considerably. From the surgeon's standpoint the reduction of the period of hospitalization is a "must" in these days when hospital beds are at a premium.

Provided the technic of transfixion is carried out precisely, there is no danger of distraction of the fractured surfaces. Healing at a normal rate is ensured because the complete and accurate fixation provides for the transmission of the line of thrust along the normal line of thrust. Early weight-bearing may be permitted following screw fixation, which helps to prevent atrophy of the soft tissues, and by increasing the circulation favors the formation of callus. Moreover, the patient's morale is improved when early weight-bearing is possible.

The drawback to screw fixation lies in subjecting the patient to the danger of infection incident to operative treatment. This risk is negligible if the surgeon is competent and provided the operation is carried out in a well equipped and well organized hospital. No sepsis was encountered in the forty-two cases being reported.

INDICATIONS FOR INTERNAL SCREW FIXATION

In the author's opinion screw fixation is not to be recognized as the routine primary treatment for all fractures of the long bones. In general, operative indications are recognized in fresh oblique, spiral or comminuted fractures of the simple or compound type in which manipulative treatment would be ineffective. These are cases in which the serrations of the fractured ends do not permit locking of the fragments or in which strong muscle forces tend to maintain the fragments in displacement. In such cases screw fixation should be carried out as the initial method of treatment and not reserved as a method of last resort, because the delay in its use may jeopardize the chances of a favorable outcome.

Screw fixation is indicated when manipulative or traction methods have been tried and failed. It is a satisfactory method of treating fractures of long standing that remain ununited or malunited following the use of other methods.

The compound fracture in which a débridement leaves the fractured area widely exposed presents ideal conditions for carrying out a transfixion. If the surgeon is competent and chemotherapy is used, early internal fixation in the open wound is a safe procedure. Union tends to take slightly longer in compound fractures than in closed injuries treated by screw fixation.

Special indications for screw fixation may be presented by certain cases that might likewise be handled satisfactorily by manipulative measures. In fractures of the shaft of the humerus the complicated retentive dressing or apparatus that must be worn following manipulative reduction is uncomfortable for an obese patient. Operative screw fixation, which requires only a minimum of external support, has special application to such cases.

Some patients prefer operative treatment in order to shorten the period of hospitalization. For instance, in the series of cases herewith reported there was a patient who had been injured far from home and it was naturally his desire to be ambulatory as soon as possible.

Special indications for operative fixation were presented in an unusual case in the series being reported. The fracture was situated in the upper part of the femur and the patient's leg on the same side had previously been amputated above the knee. Screw fixation was necessary to hold down the distal fragment which in its position of displacement upward and medially would
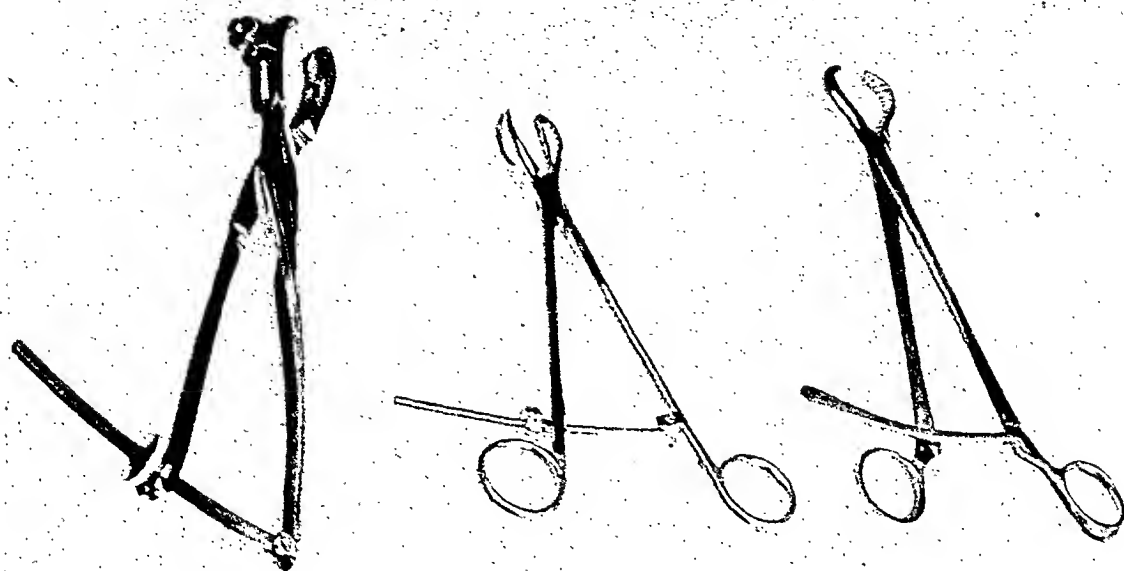
Fig. 1. Clamps used to hold fragments in position while screws are introduced.

prevent the patient from wearing an artificial leg.

It is understood that the general surgical indications of age, physical and local conditions must be favorable. The presence of blebs is not a contraindication to screw fixation even when the incision must be made in the bleb area. The formation of blebs, as well as swelling, may be prevented to some extent by bandaging the limb immediately after the injury. Screw fixation is not contraindicated in the child.

OPERATIVE PROCEDURE

Time of Operation. In closed fractures the operation is best performed within one week of the injury and the optimal time is four days. In the interval the original hemorrhage will have become stationary, the blood channels sealed over and the circulation stabilized. Also, an opportunity is provided to evaluate the damage to the soft tissues, nerves and vessels.

In compound fractures a débridement, followed by reduction and fixation, should be carried out immediately after the fracture.

Technic. The operation is carried out under rigid aseptic technic. In the simple fracture, after the skin has been prepared and the limb draped, a skin incision adequate to provide good exposure is made over the fractured area. In the case of a compound fracture a thorough débridement is first carried out.

The muscles are separated by sharp dissection, wherever possible, and allowed to fall back. Care is taken not to traumatize muscles that are split and engorged with blood. Wet gauze is used under retractors. The nerves and vessels in the proximity of the operative area are visualized and retracted if necessary. The periosteum of the main fragments is split and stripped subperiosteally wherever possible, care being taken to preserve all soft tissue attachments.

The pointed ends of the fragments are dulled in order to facilitate the approximation of the bones and to avoid damaging the soft parts. The danger of tearing gloves is also avoided by blunting the fractured ends. The fracture is reduced and the fragments are held in position by means of one or more special clamps. (Fig. 1.)

The mechanical problem is then studied. The screws when introduced should engage an equal amount of bone in each fragment as well as the cortex of each fragment. Fragments must be carefully approximated to ensure the transmission of the thrust along the normal line of thrust. The screws must be so placed that they lie as nearly as possible at the right

FIG. 2. Roentgenogram showing the distraction that may occur when screws are not placed properly.

angle to the line of fracture, thereby taking care of the stress in different directions.

The proper placing of the screws is extremely important. Figure 2 illustrates a case in which distraction occurred as the result of poor technic. The conformation of the fragment ends in this case was such that when the screws were tightened the fragments spread apart, causing distraction with subsequent delayed union. Distraction may also occur or a twisting force may give rise to absorption in the area around the screws, if the fixation material is not so introduced as to offset the stress in all directions.

One or more drill holes are made from one cortex to the opposite cortex. Screws of a non-electrolytic metal, of the self-tapping type, and of correct length are selected. The screws are seated and they should fit tightly. If the fracture is comminuted, each fragment with its attached soft tissues left intact is fixed to the main fragment by means of a screw. These fragments act as grafts. It is a mistake in treating comminuted fractures to remove a fragment, thereby creating a loss of bone substance that can make for hemidistrac-

tion. When a screw lies near the surface, as on the crest of the tibia, it should be countersunk. After the fixation has been completed the fragments should be rigid in position.

Sulfa drug crystals are dusted into the wound in cases of extensive trauma or when the fracture is compound. The wound is closed in the routine manner. The removal of the screw after union has taken place is optional except in the case of tibial fractures in which, if feasible, the fixation material should be removed.

The retentive dressing varies with the site of the fracture. In fractures of the humerus only a light retentive dressing is applied. In fractures of the femur or tibia and fibula there is need of supplementary fixation, because the threads of the screws in themselves do not provide for stability on movement of the limb in the early stage of callus formation. A thinly padded, circular cast is usually applied. If there is evidence of infiltration of the soft tissues, one or two molded plaster shells may be used first and later replaced by usable plaster dressings.

Weight-bearing with protection is started as soon as possible after the reduction of the fracture. The exact time of weight-bearing depends to a great extent upon the condition of the musculature and upon the patient's dexterity in using walking aids.

The plaster cast is changed in two or three weeks and again as required until union is complete. Care must be taken to detect and correct any angulation which would tend to delay union. Roentgenograms are taken at intervals to determine the stage of union.

SCREW FIXATION OF FRACTURES OF THE TIBIA AND FIBULA

Oblique, spiral and comminuted fractures of the tibia or of both bones of the lower leg are among the most common fractures of the long bones. The treatment of fractures of both bones is actually a problem of reducing and maintaining the tibial fragments, following which the fibu-

lar fragments fall into place. In adults the treatment of fractures of the lower leg is a problem, not only because of the difficulty of maintaining reduction but also because there is a sluggishness in the healing process, particularly in fractures of the mid and lower thirds of the tibia. As a rule, the length of time required for union to take place is entirely out of proportion to the severity of the injury.

It is customary to overcome the tendency to displacement by the use of either balanced suspension traction or double pin traction. Operators have been none too well satisfied with the results of these methods and have concluded from investigations that the establishment of union is unduly prolonged under traction methods, even when there is no apparent distraction force present. When there is obvious distraction, union is markedly delayed; for in the tibia, where the blood supply is poor, distraction has a disastrous effect. From six to twelve months may be required for union to take place. The distraction may be due to overpull in an attempt to secure precise apposition of the fragments. In fractures of both bones of the leg, when the fibula unites faster than the tibia, distraction of the tibial fragments may result from the by-passing effect of the thrust at the point of the fracture.

There is no question that the long period of hospitalization required by balanced traction is an objectionable feature. There is also the danger of angulation of the fragments under treatment by traction as well as the danger of forward or backward bowing when double pin traction is used.

Operative reduction and direct fixation is the method of choice in treating spiral, oblique and comminuted fractures of the lower leg. Several operators (Ronald, Headings, Burns and Young, Watson-Jones and Coltart, Hudack, Mansfield and Murray), after making comparative studies of fractures treated by internal fixation and by traction, reached the conclusion that the former is the preferable treatment. It offers a simpler course and is productive of good

results in a shorter period of time. The findings in the short series of sixteen fractures of the tibia and fibula being reported bear out this observation.

Analysis of Cases. Details as to the ages of the patients and the type and site of the sixteen fractures are given in Table I. Five of the fractures were compound. Fourteen were fresh fractures, in twelve of which screw fixation was carried out as the primary treatment. In the other two fresh fractures manipulative treatment had been unsuccessful. The patients in the two cases of long standing had been treated by manipulation and traction methods, and distraction had been a factor that delayed union in one patient. (Figs. 3 and 4.)

The average stay in the hospital in thirteen of the fourteen cases of fresh fractures was nine days. In the other fresh fracture the prolonged hospitalization was due to an associated fracture of the pelvis.

Union was considered to be complete when the patient was able to bear weight without the aid of retentive or supportive apparatus, was free from pain and when roentgenograms showed a continuity of callus. In estimating the average time of union, three cases were deleted from the series because one patient died from a coronary thrombosis in the third post-operative week, and in the other two cases operative reduction had been carried out late. The average time of union in the remaining thirteen cases was between sixteen and seventeen weeks. In Cases 4, 6 and 16 the delay in union must be referred to poor technic which resulted in distraction in the first case (Fig. 2), angulation with absorption of the area around the screws in the second case and separation of the fragments in the third case, due to the failure to engage both cortices in the introduction of the screws. Healing was slow in Cases 13 and 14 because of the severity of the injuries. In Case 9 there had been considerable soft tissue damage at the time of the fracture which resulted in a circulatory disturbance that delayed union. Because of the variation in ages and

TABLE I

DETAILS ON SIXTEEN FRACTURES OF THE TIBIA AND FIBULA TREATED BY SCREW FIXATION

| No. of Cases	Age	Type and Site of Fracture													Associated Injuries	Treatment before Operation	Interval between Injury and Operation	Period of Hospitalization	Deaths	Complications	Time of Solid Union
		Tibia									Fibula			Compound							
		Spiral			Oblique			Comminuted													
		U	M	L	U	M	L	U	M	L	U	M	L								
1.	59			✓								✓		✓	…	…	…	1 wk.	10 days	Coronary thrombosis 2 wk. after operation	…	
2.	60									✓			✓		✓	Multiple fractures of pelvis	…	1 wk.	4 wk.	…	…	12 wk.
3.	47						✓						✓		…	…	Manipulation; traction (distraction)	4 mo.	4 wk.	…	…	10 mo.
4.	77								✓					✓	Fracture into knee joint	Manipulation; traction	6 days	5 days	…	Distraction	8 mo.
5.	19	✓												…	…	Manipulation; traction	30 days	2 mo.	…	…	13 wk.
6.	16			✓										✓	…	…	Same day	3 wk.	…	Angulation	4 mo.
7.	36			✓						✓				…	…	…	2 days	1 wk.	…	…	9 wk.
8.	50			✓						✓	✓			…	…	…	3 days	3 days	…	…	10 wk.
9.	42								✓			✓		✓	…	…	Same day	4 days	…	Circulatory disturbance	4½ mo.
10.	47			✓									✓	…	Head and rib injuries	Manipulation	1 wk.	4 days	…	…	3 mo.
11.	10								✓					…	…	…	5 days	5 days	…	…	8 wk.
12.	38			✓						✓	✓			…	…	…	5 days	1 wk.	…	…	8 wk.
13.	36			✓						✓			✓	…	Ankle joint	…	3 days	2 wk.	…	Severe injury	4½ mo.
14.	40			✓						✓				…	…	Manipulation	2 days	2 wk.	…	Severe injury	5 mo.
15.	42													…	…	…	1 wk.	10 days	…	…	10 wk.
16.	53						✓							✓	…	…	2 days	10 days	…	Separation of fragments	20 wk.
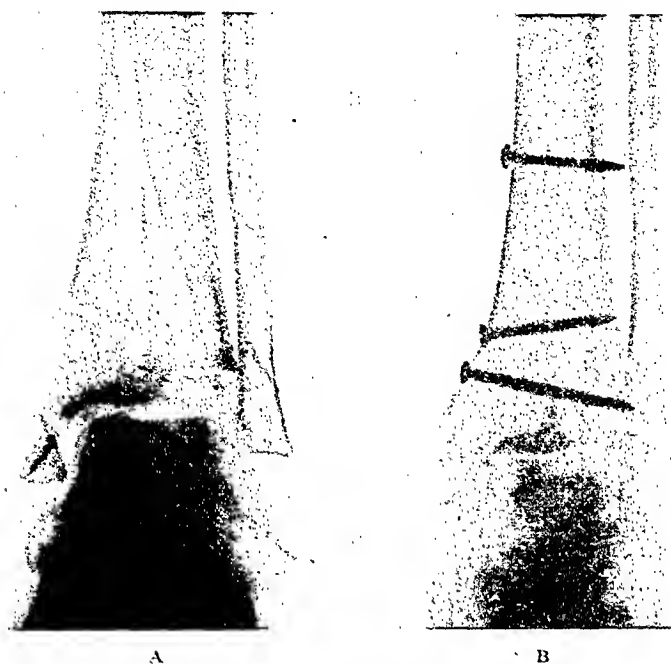
FIG. 3. Case 2 (Table 1). A, compound fractures of the lower tibia and fibula. Screw fixation was performed as the primary treatment. B, roentgenogram taken two months after operation.
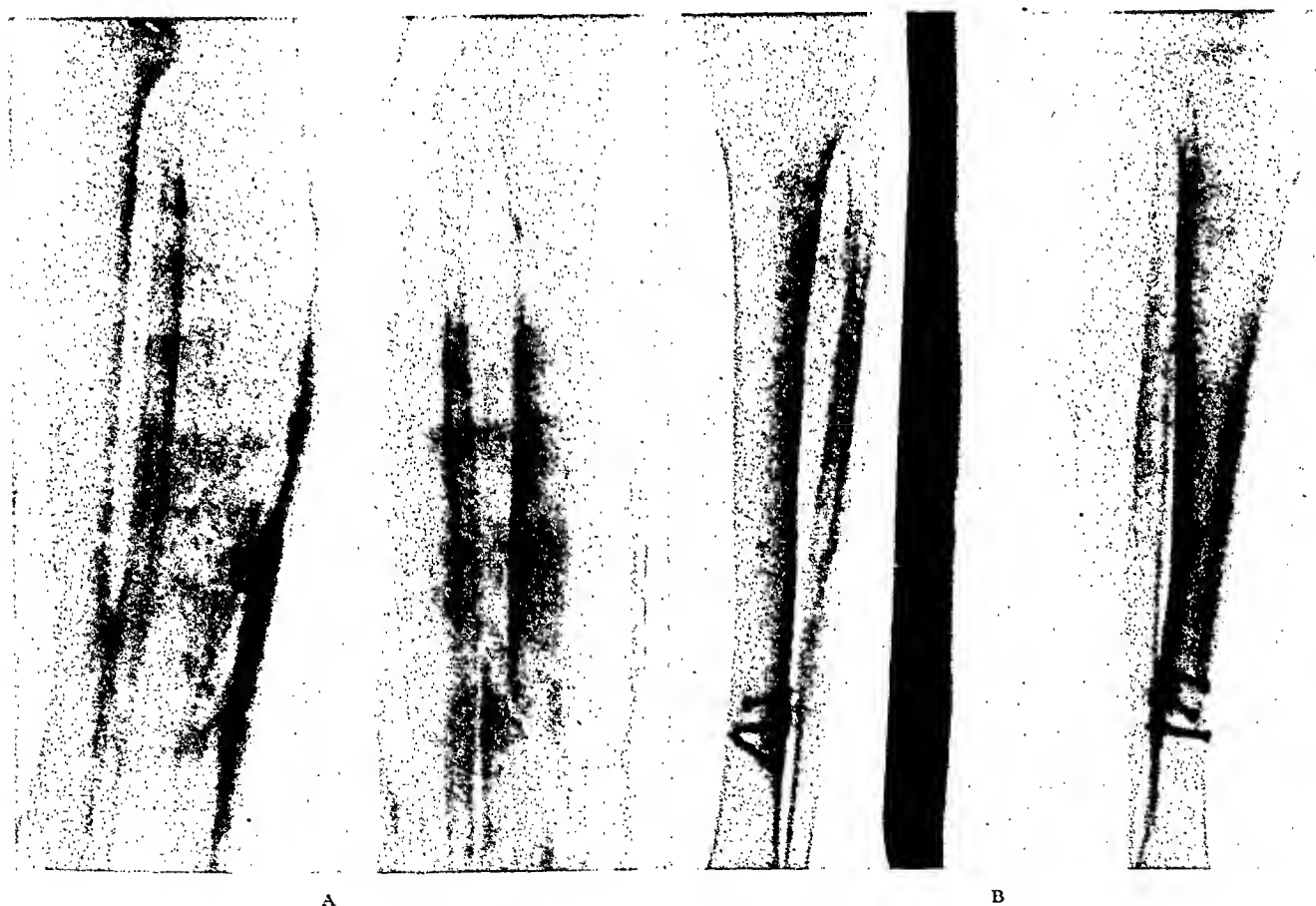

FIG. 4. Case 15 (Table 1). A, spiral fractures of the lower tibia and upper fibula with the usual displacement and posterior sagging of the tibia. Manipulative reduction proved unsuccessful. Screw fixation was performed ten days after the injury. B, roentgenogram taken six months after operation. Union took place rapidly and was solid in ten weeks.

complicating factors that influenced the recovery, it was impossible to make deductions as to whether the rate of union differed in fractures at specific sites and in different types of fractures.

All patients obtained satisfactory functional results. Details are lacking as to when patients returned to their respective occupations. One salesman (Case 9) returned to work in four weeks after reduction while the leg was still in a plaster cast. The worker in the foundry (Case 14) resumed heavy work in five months after reduction.

SCREW FIXATION OF FRACTURES OF THE FEMUR

In adults fractures of the femur are serious injuries, often entailing prolonged disability and a great time loss from work. In oblique, spiral and comminuted fractures manipulation and plaster fixation prove to be ineffective in the face of insufficient serrations of the fragment ends and strong displacing muscle forces. Particularly is this true in fractures of the upper femur in which the distal fragment is pulled medially by the forceful adductor muscle group. Under management by traction methods the average stay in the hospital is eleven weeks, and this is followed by several months of incapacity during which the limb is immobilized and protected while function is cautiously resumed (VanGorder, Funsten and Lee). It is unusual for a man to return to work in less than six months, and the convalescence often extends from ten to twelve months when a fracture is treated by traction.

In children fresh fractures above the mid-shaft may invariably be treated satisfactorily by overhead traction, but there are cases in which the corrected position cannot be maintained by this method. Overhead traction is inadequate when the fracture is low down in the shaft. Therefore, when the fracture is seated below the mid-shaft or when in fractures at any site it is evident that reduction and retention will be difficult, screw fixation is the treatment

of choice. In the child internal fixation is also indicated in fractures of the upper femur that are of long standing or malunited. In these cases in which the re-fracture necessary to effect reduction may cause severe shock, a short operative procedure such as screw fixation is particularly advisable.

The period of hospitalization following screw fixation is short, the patient usually being discharged in about two weeks. With the limb protected by a plaster cast, light weight-bearing may begin as soon as the patient is able to handle crutches well. Union in the average uncomplicated case is complete in less than four months following screw transfixion. Functionally, the results are excellent, since the danger of shortening, angulation, or impairment of joint motion is negligible.

Analysis of Cases. As shown in Table II, screw transfixion was carried out in eleven fractures of the femur. Details as to the ages of the patients and the type and site of the fractures are given in the Table. In six of the eleven cases, screw fixation was carried out as the initial treatment. In five cases the fractures had been first treated by traction methods by other physicians and had proved to be irreducible. (Figs. 5 to 7.)

The complications and associated injuries that influenced the periods of both hospitalization and recovery are listed in Table II. There was one postoperative death from severe shock in the case of a boy, aged fifteen, who had a fracture of the upper femur which was operated upon after unsuccessful treatment by traction measures for five weeks. In the five cases in which there was no associated injury or complication, the patients were discharged from the hospital in thirteen days.

The criteria used in estimating complete union were full weight-bearing capacity without pain and without external support, and evidence of continuous callus in roentgenographic studies. In nine cases (case of postoperative death and case of amputated limb excluded from the series)

TABLE II

DETAILS ON ELEVEN FRACTURES OF THE FEMUR TREATED BY SCREW FIXATION

No. of Cases	Age	Spiral			Oblique			Comminuted			Transverse			Associated Injuries	Treatment before Operation	Interval between Injury and Operation	Period of Hospitalization	Deaths	Complications	Time of Solid Union
		U	M	L	U	M	L	U	M	L	U	M	L							
1.	72			✓													6 days	12 days			13 wk.
2.	15						✓									Traction	5 wk.	15 days	Died of shock		6 wk.
3.	3	✓														Traction	18 days	21 days			26 wk.
4.	28									✓			✓		Damage to thigh muscles		5 days			One fragment irreducible	
5.	4								✓						Spine injury with foot paralysis	Traction	18 days	14 days		Foot drop	8 wk.
6.	10				✓			✓							Ankle laceration; concussion	Traction	14 days	17 days			13 wk.
7.	6																7 days	14 days			10 wk.
8.	27		✓			✓										Traction	3 days	10 days			13 wk.
9.	37			✓			✓										4 days	14 days			15 wk.
10.	52			✓						✓							6 days	13 wk.		Pulmonary infarction	30 wk.
11.	15					✓									Fracture other femur		6 wk.	13 wk.		Amputated leg on same side as fracture treated by screw fixation	26 wk.

FIG. 5. Case 9 (Table II). A, oblique, comminuted fracture of the upper femur in a patient aged thirty-seven years. Screw fixation was performed as the primary treatment. B, roentgenogram taken approximately two months after operation.

FIG. 6. Case 5 (Table II). A, comminuted fracture of the lower femur in a child aged four years. Primary treatment by manipulative and traction methods failed. Screw fixation was performed eighteen days after the injury. B, roentgenogram

FIG. 7. Case 1 (Table 11). A, comminuted, spiral fracture of the lower femur involving the knee joint in a patient aged seventy-two years. Screw fixation was performed as the primary treatment. B, roentgenogram taken three months after operation.

union was established in fifteen weeks. As was to be expected, union took place earlier in the fractures in children than in those in adults. It was not possible to make deductions as to the rapidity or delay in healing in various types of fractures or in fractures at different sites, owing to the variation in ages and complicating factors.

The functional results were satisfactory; the patients walked without a limp and with free joint motion. Details on the time resuming work were obtained in only two of the five adult cases. One farmer (Case 9) returned to work in four months after the operation. It was a year before a driver of a tractor (Case 4) resumed work, but in this case an irreducible fragment had persisted as a sensitive spur and had to be removed.

SCREW FIXATION OF FRACTURES OF THE HUMERUS

In certain types of fractures in the upper end of the humerus it is difficult to effect reduction and ensure retention of the fragments. In this group is the spiral or comminuted fracture situated below the insertion of the pectoralis major muscle, in which the upper fragment is pulled inward toward the axilla and rotated forward by the pectoralis muscle. (Fig. 8.) Indications for operative reduction and internal fixation should be recognized in this type of fracture. Following screw fixation, no complicated dressing or apparatus is necessary.

Another type of fracture of the upper end of the humerus, which usually can be reduced only by surgical intervention, is a break involving the upper epiphyseal plate
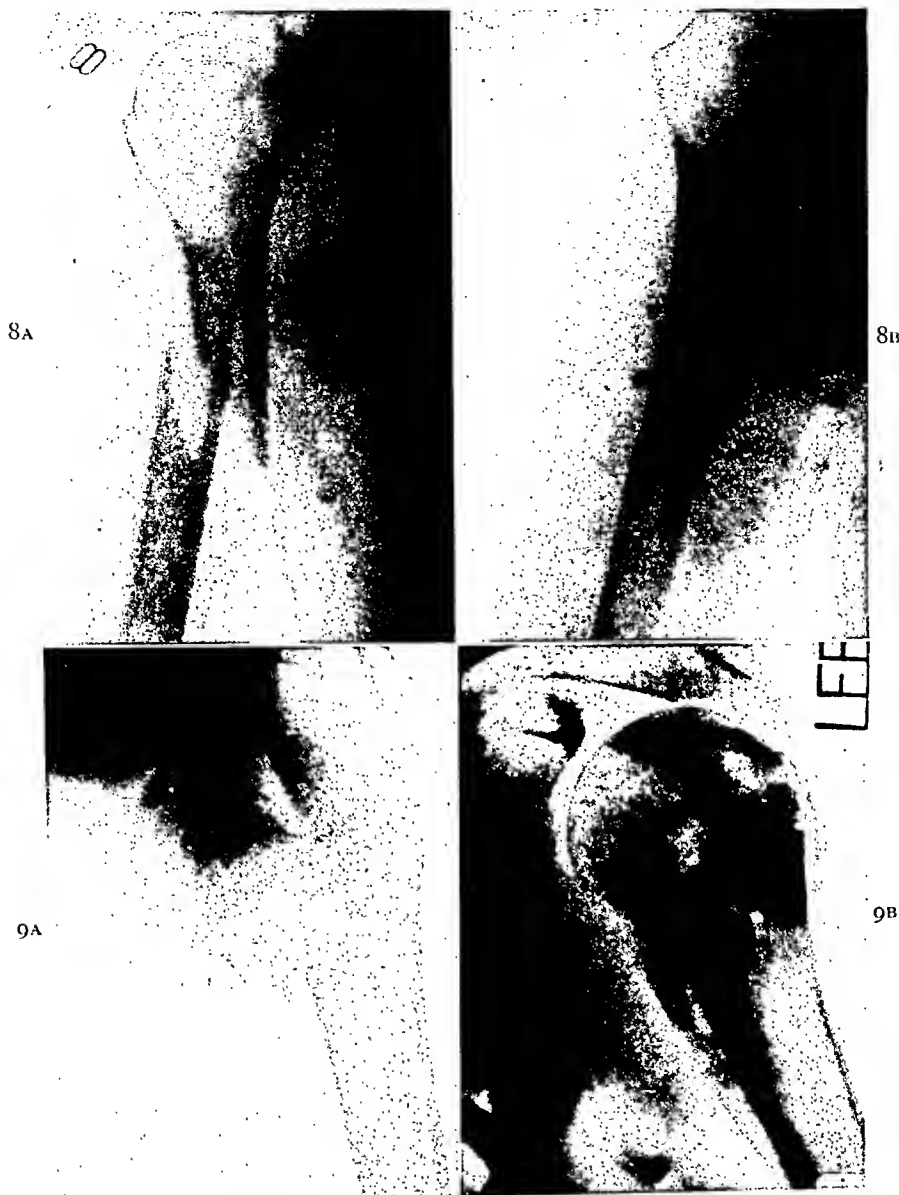
FIG. 8. Case 4 (Table III). Spiral fracture of the upper end of the humerus with adduction displacement of the proximal fragment. Primary treatment by manipulation failed. Screw fixation was performed one week after the injury. B, roentgenogram taken four days after operation.

FIG. 9. Case 11 (Table III). A, fracture of the upper epiphyseal plate of the humerus with a spicule of the shaft attached to the proximal fragment. Primary treatment by manipulations failed. Screw fixation was performed two weeks after the injury. B, roentgenogram showing the screw in place. Note the overgrowth of callus along the humeral shaft. (This illustration appeared in *J. Bone & Joint Surg.*, 26: 401, 1944.)

in which a spicule of the shaft is split off with the humeral head. (Fig. 9.) This fracture is uncommon and is seen in adolescents. Conservative methods are not only ineffective but manipulative attempts at reduction may actually be harmful in that they result in the formation of callus along the humeral shaft. This callus tends to persist even when operative reduction is finally obtained.

There are two reasons why conservative methods are inadequate in the treatment of these fractures: First, the line of fracture is slanting and irregular and the broad
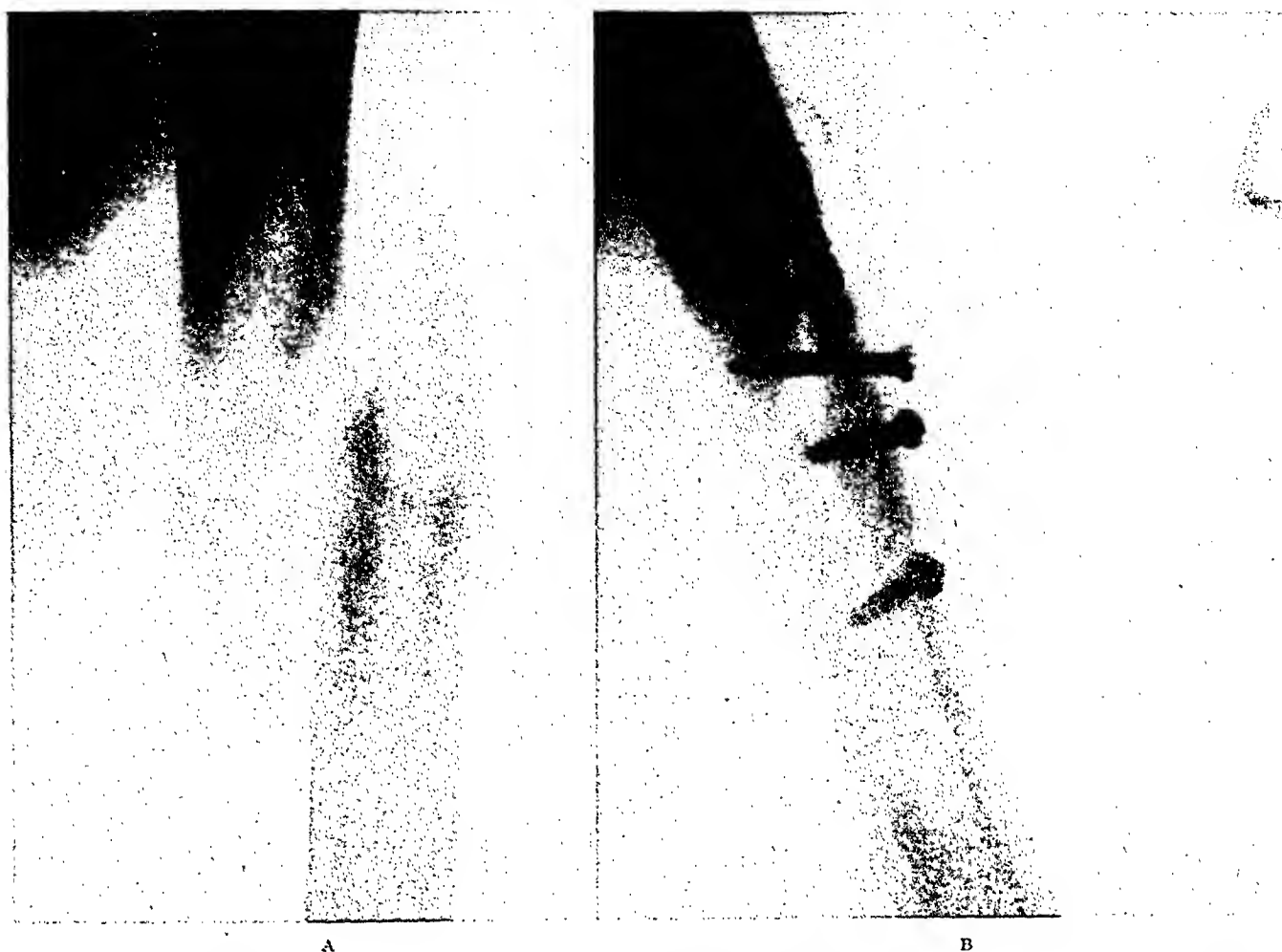
Fig. 10. Case 7 (Table III). A, comminuted fracture of the mid-shaft of the humerus, irreducible by manipulative and plaster fixation method. Screw fixation was performed five and a half weeks after the injury. B, roentgenogram taken one month after operation.

upper and outer edge of the distal fragment is thrust through a slit in the capsule of the joint and gripped tightly by the capsule; secondly, the long head of the biceps tendon lies between the fragments. When traction is applied in an attempt to reduce the fracture, both the capsule and the biceps tendon become taut, thus preventing reduction of the arm. Reduction cannot be accomplished by rotating or angulating the fragments because the tight capsule carries the head fragment along with the shaft.

When an operative reduction is carried out early before the capsular sleeve extension is infiltrated with exudate to the point of contracture, it is usually possible to suture the fragments. When, however, operative indications are not recognized for several days, there is a tendency for displacement to recur if screw fixation of the fragments is not carried out.

Fractures of the mid-shaft of the humerus seldom require internal fixation. Operative treatment is indicated in the compound fracture and in an occasional case of a closed fracture in which reduction or the maintenance of the fragments in the correct position is impossible. (Fig. 10.) Screw fixation can also be used to advantage in fractures of the mid-shaft when the patient is obese and would find the retentive dressing supplementing manipulative handling uncomfortable. Screw fixation is an excellent form of treatment when it is imperative to avoid prolonged hospitalization and convalescence.

In fractures of the lower end of the humerus with a short distal fragment, it is difficult to obtain and hold reduction by manipulative or traction methods. Accurate reduction, on the other hand, is important because any overgrowth of

TABLE III

DETAILS ON THIRTEEN FRACTURES OF THE HUMERUS TREATED BY SCREW FIXATION

No. of Cases	Age	Oblique Spiral U	M	L	U	M	L	Comminuted U	M	L	Oblique with Spicule Upper	Compound U	M	L	Associated Injuries	Treatment before Operation	Interval between Injury and Operation	Period of Hospitalization	Complications	Time of Solid Union
1.	63		✓													Traction	9 days	14 days	Weak muscles; bone soft, cortex thin	10 wk.
2.	60	✓															1 day	5 days		9 wk.
3.	55				✓												Same day	4 days		9 wk.
4.	57							✓							Periosteal tearing	Manipulation	7 days	12 days		10 wk.
5.	63																Same day	7 days		10 wk.
6.	44								✓				✓		Compound 3 places		At once	10 days	Severe damage to soft tissues	13 wk.
7.	30												✓		Compound fracture patella	Manipulation	5½ wk.	14 days		8 wk.
8.	55			✓													5 days	3 days		10 wk.
9.	49																3 days	2½ wk.		10 wk.
10.	13										✓					Two manipulations	2 days	4 days		8 wk.
11.	15										✓					Two manipulations	1 wk.	4 days		8 wk.
12.	15										✓					Repeated manipulations	2 wk.	4 days		8 wk.
13.	15										✓					Repeated manipulations	2 wk.	4 days		8 wk.

FIG. 11. A, oblique fracture of the ulna accompanied by a slight dislocation of the head of the radius that is not demonstrated in the roentgenogram. Following treatment by splinting, further dislocation of the radial head occurred. Screw fixation of the ulnar fragments and removal of the head of the radius were carried out two months after the injury. B, roentgenogram taken one week after operation.

callus may lead to involvement of the radial nerve. Screw fixation is indicated in these fractures.

Analysis of Cases. The author's series of fractures of the humerus comprised thirteen cases, two of which were compound fractures. Table III gives the details as to the ages of the patients and the type and site of the fractures. Screw fixation was carried out as the primary treatment in six cases. In six of the seven patients who had been under the care of other physicians manipulative treatment had failed, and in the seventh case traction had been unsuccessful.

The period of hospitalization was short, the average stay in the hospital in the thirteen cases being eight days. Healing was considered complete when the patient had full use of the arm and there was roent-

genographic evidence of solid union. In the thirteen cases the average period required for union was nine and one-quarter weeks. In Case 6 the delay in union was due to the severity of the injury, the fracture being compound in three places and associated with extensive damage to soft tissues. The functional results in all cases were excellent and most patients could use the arm before union was complete.

SCREW FIXATION OF FRACTURES OF THE FOREARM BONES

Short oblique fractures of the radius may present difficulties in reduction and retention. Short oblique fractures of both bones of the forearm situated at either the same level or at different levels, as in the upper third of one bone and in the mid-third of the other bone, may be difficult to control.

There is also the danger that if the ulna unites ahead of the radius, a distraction force and off-line stress may result that delays union in the radius. Another forearm fracture that is difficult to control is the short oblique or spiral fracture of the ulna accompanied by dislocation of the head of the radius (Monteggia's fracture).

Operative reduction with internal fixation of the ulnar fragments is the accepted primary treatment of the Monteggia's fracture. Open reduction is indicated in forearm fractures of other types that are difficult to control by conservative measures; and if the fragments cannot be locked in position, internal fixation is required. A single screw is an efficient form of fixation, provided the fragments are sufficiently oblique.

CASE REPORT

The patient, a man, aged twenty-five years, sustained a fracture of the ulna accompanied by a slight dislocation of the head of the radius. The fracture was treated by manipulation by another physician and a splint applied. Two months later the case came to the attention of the author.

A roentgenogram taken at the time of the examination showed a Monteggia's fracture with non-union of the ulnar fragments. A long spur projected from the ulna, producing a synosteosis between the forearm bones. (Fig. 11A.)

On May 17, 1943, an operative reduction was performed. The head of the radius was removed. The ulnar fragments were freshened and fixed in position by means of a screw. (Fig. 11B.)

Seven weeks after the operation the fracture was clinically solid, but the recovery of rotary motion was delayed because of the absent head of the radius. In May, 1946, three years after the reduction, the elbow motion was good but the rotary motion was still limited.

SUMMARY

Experience with internal screw fixation in a relatively small series of forty-two cases leads to the conclusion that the method has an established place as the treatment of selected fractures of long bones. The results were of good quality; there was no shortening, no deformity and no disturbance of joint function.

Operative screw fixation has certain advantages over the traction methods in use. It requires only a short period of hospitalization. The patient is free from pain and discomfort. Although it is not possible to make an accurate estimate of the period of recovery owing to the many circumstances and factors that have a bearing on healing, the conclusion seems justified that, in this series of cases, the time required for union was shorter than the period generally estimated for healing under traction methods.

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ETIOLOGY OF GOITER IN MAN*

NEW CONCEPTS

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THE establishment of the existence and powerful operation of whole systems of goitrogens would alone seem to necessitate a drastic revision of the familiar concepts concerning the etiology of goiter in man. But also the newer biochemical and pharmacological knowledge comprehends such novel factors as biologic or metabolic competition between structurally related compounds, including structural analogues antagonistic to thyroxine. In accordance with clear scientific logic, the possibly basic causative action of a variety of chemical influences hitherto unknown or neglected must be taken into consideration in work upon the problem of goiter etiology. The purpose of this paper is to indicate the *possibilities* that must be accounted for by adequate researches rather than to speculate upon *presumable probabilities*. Justification for such a discussion of possibilities is found in the fact that the problem of goiter causation has not previously been considered in the light of the newer and apparently revolutionary findings in regard to goitrogens and metabolic competition between similar and, in certain instances, dissimilar molecules, especially thyroxine and related compounds.

GOITROGENS

The existence of goitrogens was suspected for centuries. Innumerable early writers suggested that some chemical cause of goiter, especially endemic goiter, would be discovered in drinking water or certain articles of diet. In many goiter regions the comparatively high concentrations of calcium in the water have repeatedly attracted attention to this element as a possible etiologic factor. Excess of pork or fat was long suspected, as by St. Lager (1867). Indeed it was shown by Marine and Lenhart (1910) that thyroid hyperplasia may be induced in dogs and brook trout by the feeding of pig's liver. McCarrison (1920) reported that fat-feeding had a similar effect in pigeons; Mellanby and Mellanby (1921) also brought about thyroid hyperplasia in dogs by the same means.

Bircher (1911) stated that he was able to induce the appearance of large goiters in white rats by giving them water from a region in which goiter was endemic; further, these animals showed great enlargement of the heart with hypertrophy of the left ventricle. Hellwig (1944) expressed the belief that a high calcium intake may indeed be the cause not only of goiter but of "goiter heart"; and his experimental findings on white rats fed a high-calcium diet were similar to those reported by Bircher to follow administration of "goiter-causing" water. No confirmation of such work has been forthcoming.

Other lines of investigation have been vastly more profitable and the results are now indisputable. Numerous goitrogens have been shown to be present in common foods of animals and man, and systems of compounds having goitrogenic activity have been found among familiar or newly synthesized drugs.

In 1928 Chesney, Clawson and Webster noted that a colony of rabbits were rapidly becoming goiterous; it was soon established that the etiologic factor was present in cabbage. The thyroid glands were extremely hyperplastic but the animals had low metabolic rates. Iodine administration was found to prevent the development of "cabbage goiter," but when iodine was

given after the goiter had developed, a severe thyrotoxicosis resulted promptly. These findings were confirmed by Marine, Baumann, Spence and Cipra (1929, 1932) who determined also that the leaves of many brassica plants contained some goitrogen. Suk (1931) reported the endemic occurrence of large goiters in a community in Carpathian Ruthenia where the principal dietary item was cabbage.

Because of the reported isolation of certain nitrile compounds from the leaves of several brassicae (cabbage, Brussels sprouts, cauliflower), the action of a number of cyanide compounds on the thyroid glands of laboratory animals was investigated by Marine and his associates (1932, 1934), Spence (1932, 1934) and others. It was observed that methyl cyanide and certain other nitriles produced marked thyroid hyperplasia with low metabolic rates and exophthalmos. Sharpless et al. (1939) recorded the production of goiter in rats by feeding raw or treated soy bean flour; such goitrogenic action was prevented by administration of iodine. In 1941 Kennedy and Purves found that rape seed and the seed of other brassicae can be used to produce goiters in rats.

Mackenzie, Mackenzie and McCollum, also in 1941, found that when sulfaguanidine was fed to rats for short periods of time, there resulted a marked enlargement and hyperplasia of the thyroid gland, an effect that was not inhibited by iodine. Richter and Clisby (1941, 1942) noted that phenylthiourea was goitrogenic, and, at about the same time, Kennedy, Purves and Griesbach described in greater detail the goitrogenic action of rape seed, Kennedy later (1942) attributing this action to allyl thiourea, presumably a constituent of rape seed. The concepts that goiter resulting from the action of sulfonamides and thiourea is compensatory and that the primary effect of such substances is inhibition of synthesis of thyroid hormone were set forth by the researches of the Mackenzies (1943) and Astwood and his associates

(1943). Astwood and others followed up the suggestion that compounds related to the sulfonamides and thiourea might be valuable therapeutic agents. The chemical nature of the compounds which inhibit the function of the thyroid gland was discussed by Astwood (1943) who at that time remarked that the compounds tested could be divided into three classes: thiourea derivatives, those containing an aminobenzene grouping and the thiocyanates. The thioureas as a group are the most active goitrogens, thiouracil showing the highest activity. The aminobenzene derivatives include the common sulfonamide drugs, of which sulfadiazine is the most active. Iodine administration prevents the goitrogenic action of the thiocyanates but not that of the other two classes.

The differentiation of the antithyroid action of thiouracil, thiourea and para-aminobenzoic acid from sulfonamides by iodine administration has very recently been described in detail by C. G. Mackenzie (1947). Mackenzie observed that small amounts of iodine inhibit the goitrogenic action of thiouracil by 50 to 100 per cent in rats, depending upon the level of the goitrogen employed. Higher levels of iodide do not increase this inhibition but suppress thyroid hyperplasia. When a low level of sulfaguanidine is administered, the goitrogenic effect is augmented by a high level of iodide; but this augmentation, or synergistic action, is not noted with either low levels of iodine or a high level of sulfaguanidine. Nevertheless, according to Mackenzie, iodide seems to increase the degree of thyroid hyperplasia under all the conditions studied. Further, para-aminobenzoic acid resembles thiouracil rather than sulfaguanidine insofar as both its goitrogenic and hyperplastic actions are inhibited by iodide. Mackenzie concluded that thiouracil, thiourea and para-aminobenzoic acid differ from the sulfonamides in the primary biochemical reactions responsible for their effects on the thyroid gland. Thus it is apparent that considerable modification of

earlier views concerning the relation of iodine intake to goitrogenic action is necessary. Depending upon the iodine level and upon the goitrogen, the goitrogenic action is either intensified or inhibited.

Other fundamental differences between goitrogens have been brought to light. Rawson, Tannheimer and Peacock (1944) employed a radioactive isotope to demonstrate that the thiocyanate goiter readily takes up iodine whereas the enlarged glands resulting from thiouracil administration do not. Species difference is still another highly important factor. When administered to chicks, aminobenzene derivatives show no goitrogenic effect (Mackenzie and Mackenzie, 1943), whereas in this species thiourea derivatives have high goitrogenic activity (Astwood et al., 1944; Mixner et al., 1944.)

It is well established that administration of desiccated thyroid or thyroxine inhibits the action of goitrogens in general (Webster and Chesney, 1928; Marine et al., 1932; Astwood, 1943; Salter et al., 1945). Griesbach, Kennedy and Purves (1941) showed that the pituitaries of animals having goiters produced by a brassica seed diet present histologic changes similar to those observed in the pituitaries of thyroidectomized animals. These investigators reported also that hypophysectomy prevents the development of goiters in animals which later receive goitrogens. Hence there arose the concept that the action of the various drugs which produce goiter is by means of an inhibition of, or interference with, the manufacture of the normal thyroid hormone (Rawson, Hertz and Means, 1943; Franklin and Chaikoff, 1943; Salter et al., 1945). The resulting increase in activity of the thyrotropic hormone of the pituitary induces hyperplasia of the thyroid cells, thyroid hormone production being at the same time decreased rather than increased. These investigations eventually led to the introduction of goitrogens into therapy of hyperthyroidism (Astwood, 1943; Himsworth, 1943; Williams and Bissell, 1943).

GOITER PRODUCTION IN MAN BY GOITROGENS

Hundreds of compounds have been tested and found goitrogenic and toxic in varying degrees when administered to animals. Many have been and are being used extensively (if not indiscriminately) in the treatment of thyrotoxicosis and even non-toxic adenomatous goiter. It is obvious, therefore, that many different agents not only may cause goiter in man but are actually being therapeutically employed as a result of their recognized goitrogenic action.

Goitrogenesis as an undesirable side-effect of a number of chemotherapeutic agents has also become a familiar manifestation. Goiters develop in a certain small percentage of hypertensive patients who are being treated with thiocyanate (Barker et al., 1941; Fahlung, 1942; Kobacker, 1942; Foulger and Rose, 1943; Rawson et al., 1943). The observations of Rawson and associates are typical. Goiters appeared in two patients who were receiving potassium thiocyanate for treatment of hypertension. Decreased thyroid function was indicated by clinical symptoms of hypothyroidism, low basal metabolic rates and plasma protein iodine levels usually observed in myxedema. An extreme hyperplasia of the thyroid gland was demonstrated in biopsy material removed from one patient. In one case there was exophthalmos with lid lag. In both cases the goiters and clinical signs of hypothyroidism disappeared when thyroid was administered in conjunction with thiocyanate.

A number of cases of goiter associated with sulfonamide therapy have been recorded, and related compounds have also been shown to be goitrogenic. In fact, goitrogenesis must now be recognized as an occupational hazard. Perrault and Bovet (1946) stated that in 1943 Jeantet of Lyons, France, observed unusual goiters in workmen employed in the extraction of aminothiazole which is used in the preparation of sulfathiazole. Perrault and Bovet were sufficiently impressed by the goitrogenic

activity and lack of toxicity of amino-thiazole to try it in therapy of hyper-thyroidism. The results in a large group of patients convinced these workers that the goitrogen is more effective and much less toxic than thiouracil.

Rawson, Hertz and Means (1943) have remarked that the action of goitrogens must be regarded as a problem of practical importance in view of "the advocated use of soy beans in the modern diet, the liberal prescribing of the sulfonamides in clinical medicine and widespread use of thiocyanate in treating hypertension."

Recent research suggests additional possibilities that have practical aspects. The very number of different substances already shown to be goitrogenic is remarkable and may not have received the attention it would seem to deserve. The extreme *lability* of the thyroid gland, long recognized as the most labile organ in the human body, is thus once again made manifest. Hundreds of substances have been proved goitrogenic when ingested in food or water. Many of these substances may be regarded as drugs from one point of view, but from another they are food factors, some indispensable, as in the instance of para-aminobenzoic acid. Many common foods contain goitrogenic substances; this fact is now beyond question.

Obviously, the ancient speculation that goitrogens would be found in foods has been transmuted into established fact. The fact is highly suggestive of possibilities, many of which would seem to be eminently worthy of practical consideration.

The number of *classes* of goitrogens is being steadily augmented. A striking addition to the list of known goitrogens is selenium. In 1946 Seifter and associates reported that the thyroid glands of white rats show increase in size, hyperplasia and loss of colloid after ten days on a diet containing 0.05 to 0.1 per cent of an organic selenium compound (*bis*-4-ace-tamino-phenyl-selenium). After 105 days on a diet containing the selenium compound at the lower level, the animals develop multiple adenomas of the thyroid gland.

As is well known, selenium occurs in toxic concentrations in the soil, water and vegetation of areas in at least thirteen States. More than 100,000 acres of seleniferous farmland have been taken out of cultivation in recent years. Nevertheless, many areas whose soil and water contain selenium (at a comparatively low and presumably non-toxic level) still are under cultivation. The significance of the low content of selenium in wheat grown in such seleniferous soil remains to be determined. The significance of goitrogenic agents in cabbage, cauliflower, other edible brassica plants, and in soy beans is apparently greater, and is generally believed, perhaps correctly, to have no relationship to endemic goiter. Possibilities can be eliminated only by research.

Investigations upon the effects of goitrogens ingested by man must henceforth be guided by the knowledge that species differences are at times marked. Another complicating factor is the possible synergism between two or more different goitrogens received at the same time and between iodine (at a certain level) and one or more goitrogens (Mackenzie, 1947). Moreover, now that the existence of many antithyroid substances has been established, the possibility is suggested that there may be groups of substances whose primary "target organ" is the pituitary, the thyroid gland being secondarily affected. Synergism between antipituitary and antithyroid substances—in varying combinations and concentrations—must be placed on the list of possibilities.

STRUCTURAL ANALOGUES ANTAGONISTIC TO THYROXINE

The discovery of biologic and metabolic competition between structurally related compounds has incited extensive biochemical researches whose findings already constitute a great new division of physiologic chemistry (Woolley, 1947). Within the past few years there have been found in nature or synthesized in the laboratory compounds which are structurally similar

to, but biologically antagonistic to metabolically important substances. These agents compete with hormones, vitamins or other metabolites and manifest their effects by the production of signs of deficiency of the metabolite to which the "bio-antagonist" is related in specific structural ways. For instance, in the testing of the presumed vitamin-like activity of substances structurally related to thiamine, it was found that instead of exhibiting thiamine-like effects certain structural analogues of thiamine induce signs of vitamin B_1 deficiency when the laboratory animal receives the "bio-antagonist" *and* thiamine (at a level otherwise high enough to prevent the appearance of signs of deficiency).

Because many "bio-antagonists" of metabolically important substances have been found to produce signs of deficiency of the analogous metabolite, Woolley proposed that a new series of pharmacologic agents might be produced by altering the structures of hormones or vitamins in definite ways. According to Woolley: "It was considered possible that such inhibitory structural analogues could be found against certain hormones which, as a result of overproduction, or diminished rate of destruction in the body, are the causative agents of disease. The excess hormone might be counterbalanced by administration of the structurally related antagonist." Woolley (1946) has been able to synthesize several new ethers of N-acetyldiiodotyrosine (structural analogue of thyroxine) that have been found to counteract the pharmacologic effects of thyroxine in experimental animals.

Investigators interested in the etiology of goiter must now consider the fact of the existence of structural analogues of thyroxine that counteract the effects of the hormone. Such analogues, "bio-antagonists," may exist in foods or may be produced normally or pathologically in the body.

Because thyroxine inhibits the action of many if not all goitrogens, *a priori* it would seem that the administration of goitrogen in conjunction with a bio-antagonist of

thyroxine would be expected to enhance markedly the action of the goitrogen. (It is to be noted that certain of the bio-antagonists of thyroxine which were synthesized by Woolley have at once a strong antagonistic action (antithyroxine properites) and a weak thyrosine-like activity. The antithyroxine substance when administered alone shows weak thyroxine-like activity; administered in conjunction with thyroxine, the bio-antagonist signally diminished the effects of the metabolite. Goitrogens do not show thyroxine-like activity).

The existence of antihormones has long been postulated. Antithyroxine bio-antagonists are antihormones.

IODINE DEFICIENCY AND ENDEMIC GOITER

In 1916 Marine and Kimball (1917) instituted the prevention of endemic goiter in man by iodine prophylaxis on a large scale through the public schools of Akron, Ohio. By 1920 it had been demonstrated convincingly that the incidence of endemic goiter in adolescent girls could be reduced to a very low figure by keeping the thyroid saturated with iodine (Marine and Kimball, 1920). This brilliant and convincing demonstration of the definite relation between iodine deficiency and endemic goiter resulted, as is well known, in the almost universal adoption of iodine prophylaxis as a public health measure in goiter regions. The successes have been impressive. For instance, in 1924 a survey showed that the incidence of goiter in certain goiter sections of Michigan was 38.6 per cent. Iodine prophylaxis (by iodized salt) reduced the incidence to 8.2 per cent. In Cleveland the incidence of goiter among school children was found to be 31 per cent in 1924; iodine prophylaxis brought the incidence down to 18.5 per cent in 1937; the incidence among non-users of iodized salt was 30.7 per cent in 1937, among users of iodized salt, 7.7 per cent (Kimball, 1939). Because of neglect of this definitely indispensable public health measure, goiter has been on the increase in a number of goiter sections (Kimball, 1946).

Because of such great successes resulting from the large scale use of iodine prophylaxis in endemic goiter regions, many authors have promulgated the conclusion that iodine deficiency is the cause of all cases of endemic or simple goiter. Nevertheless, there are many facts which such a conclusion does not take into account. Iodine prophylaxis is effective in most but not all cases. Kimball (1939, 1946) stated that in certain sections of Michigan the incidence of goiter "among those using iodized salt regularly" was 2.88 per cent in 1936, twelve years after the institution of iodine prophylaxis in these sections. In Cleveland in 1937 the incidence of goiter among school children who were users of iodized salt (presumably since early infancy) was 7.7 per cent. It is a fact that iodine administered at a comparatively high level does not *completely* solve the problem of endemic goiter; children presumably "saturated" with iodine—certainly receiving iodine at a higher level than the average individual in many regions where goiter does not occur—nevertheless show a strikingly high incidence of goiter.

In a recent editorial on progress and future in the treatment of goiter, Cole (1944) stated: "It must be emphasized that restoration of the proper iodine intake alone cannot be expected to eliminate goiter because goiter of all types still exists along the seaboard where iodine intake (particularly in drinking water) is presumably adequate. Moreover, the added intake of iodine in iodized salt in these communities during the past few years has not resulted in any decrease in goiter. As a matter of fact, in some communities (for example, New Orleans) the incidence of goiter appears to be on the increase."

The incidence of goiter in parishes (the counties of other states) in the southern part of Louisiana, where iodine intake appears to be well above the average for the country at large, has impressed a number of authors. Mahorner (1944) has remarked

in a recent paper: "Goiter is not uncommon among peoples inhabiting the banks of certain bayous. These rivers, often very deep, were originally estuaries of the Mississippi River. The surrounding lands are flat and low and frequently marshy and were formed by alluvial deposits of the Mississippi River. The diet of the people inhabiting this area consists presumably of much sea foods, and the iodine content of their vegetables is relatively high. The people themselves along these bayous recognize that nodular goiters are common. The explanation as to why the people in this low flat area should have more goiters than those in the rest of Louisiana is not available. All the soil of this area was brought down the river possibly from the upper Mississippi and the Ohio Rivers, which drain goiter belts. A positive goitrogenic factor thus may have been transported."

There are additional evidences that deficiency of iodine cannot account for all cases of simple goiter. Sporadic goiter occurs in regions where the iodine intake is high. "Epidemics" of goiter have been known to occur, as remarked many years ago by Hirsh (1883); these must have been evanescent exacerbations of general conditions among a population already affected by deficiency of iodine, but the iodine deficiency does not explain the sudden increase in incidence of endemic goiter. And now, of course, the production of goiters deliberately (in therapy of hyperthyroidism by administration of goitrogens) and inadvertently (by thiocyanates, sulfonamides and related compounds) has become a familiar demonstration of goitrogenesis without iodine deficiency.

ETIOLOGY OF THYROTOXICOSIS

In experimental animals severe thyrotoxicosis can be induced by (1) rendering the animal goiterous through the action of any one of variety of goitrogens and (2) administering iodine. Similar factors *may* be operative under natural conditions.

This possibility should interest the "unitarians" especially, i.e., those who have long maintained that simple goiter, exophthalmic goiter and toxic adenoma have the same underlying pathology. Until more has been determined concerning the action of goitrogens under natural conditions, discussion would be mere speculation. In the meantime, we may wonder about apparent relevancy of the fact that iodine administration occasionally causes exacerbation of Graves' disease and may even, as many have asserted, precipitate Graves' disease if relatively high dosages are given to patients with simple goiter. Harington (1933), for instance, has pointed out: "Graves' disease was not at that time recognized, but the description by Gairdner (1824) of the condition of goitrous patients after overvigorous treatment with iodine leaves no doubt that the syndrome of Graves' disease had been elicited or at least precipitated in them by the use of this drug."

ADENOMATOUS GOITER

Gorbman (1946) has shown that prolonged feeding of a goitrogen (thiourea) to mice causes the development of adenomatous goiters. Presumably these "non-toxic adenomatous" goiters could readily be transformed into "toxic adenomatous" goiters by administration of iodine although Gorbman has not mentioned the possibility. It is to be noted, however, that the goitrogen was fed at an extremely high level: thiourea constituted 2 per cent of the diet of the animals. (No approximation to such a level is ever attempted in therapy with antithyroid substances.)

METASTASES OF THYROID TISSUE

Gorbman (1946), after prolonged feeding of thiourea to mice, detected thyroid cells in veins of the thyroid gland, these cells evidently having become detached from the highly activated epithelium. This finding led him to examine the lungs wherein he was able to demonstrate histologically the presence of small islands of thyroid-like cells showing high mitotic activity. (Resumption of the normal diet was followed by involution of the activated epithelium.) The significance of these observations in relation to the possible natural action of goitrogens during a period of years cannot now be surmised. The possible co-action of a carcinogenic influence would be another consideration.

TOXIC AND NON-TOXIC ADENOMATOUS GOITER

The demonstration of the existence of structural analogues antagonistic to thyroxine, i.e., "bio-antagonists" of thyroxine, suggests the possibility that the presence of some such antihormone may explain certain differences between toxic and non-toxic goiter. If the bio-antagonist were present in non-toxic adenomatous goiter, presumably at least some of the effects of thyroid hormone would be counteracted. Disappearance of the biological competitor would, perhaps, explain the development of toxicity in a previously non-toxic case. A complicating factor would be fluctuating iodine intake.

"CARDIOTOXIC" GOITER

As early as 1887 Schranz remarked the occurrence of cardiac abnormalities in patients with goiter, especially adenomatous goiter, but without other symptoms or signs of a toxic condition. Among the many authors who have expressed a conviction that there is such an entity as "cardiotoxic" goiter are Hertzler (1936), Schmidt and Hertzler (1942), Rasmussen (1941) and Meyer and Ferguson (1942). Of course, as is well known, congestive heart failure may result from thyrotoxicosis alone (Rose, 1946). Plummer (1921) suggested that the thyroid gland in certain thyrotoxic patients secretes an abnormal thyroglobulin-like substance. Rose (1946) remarked: "This suggestion may have been too readily abandoned." Many authors have theorized that the heart may be directly affected by a specific "cardiotoxic" agent elaborated by

a goiter, whether or not the basal metabolic rate is elevated.

The discovery of thyroxine analogues antagonistic to thyroid hormone indicates still another possibility: Certain bio-antagonists may be *modulators* of the effects of thyroid hormone rather than *direct* antagonists. In fact, Woolley (1946) presented no evidence to show that *all* effects of thyroxine are directly counteracted by the antagonistic analogues tested. A modulator of thyroid hormone activity would, presumably, counteract or completely neutralize certain effects of the metabolite whereas other effects might even be augmented by a partial synergism. It is even conceivable that the physiologic effects of a metabolite might be given a new pitch or "keynote" so as to have the total influence of the metabolite exerted in a new, single direction toward some target organ such as the heart. It has not yet been demonstrated that any bio-antagonist is not a modulator rather than a direct antagonist. It is to be noted that however great the quantity of bio-antagonist present in the body, the metabolite is still present, capable of playing new rôles if not the old.

SUMMARY AND CONCLUSIONS

1. The demonstration of the existence of powerful goitrogens, many of which occur naturally, would seem to open the way to new concepts in the investigation of the causative factors in simple or endemic goiter.

2. Iodine deficiency is the outstandingly important causative factor in the development of simple goiter, but other factors are important also.

3. It is possible that goitrogens in drinking water, common foods or both are involved in the etiology of simple goiter.

4. New etiologic concepts concerning goiter are also suggested by the demonstration of the existence of structural analogues of thyroxine that counteract or modulate the effects of thyroid hormone.

5. Goitrogens and perhaps also bio-antagonists of thyroxine may be involved in the causation not only of simple goiter but also of exophthalmic goiter, toxic adenoma and non-toxic adenoma.

6. Under certain conditions prolonged ingestion of goitrogens may eventually cause the production of metastasizing tumors of the thyroid gland.

7. "Cardiotoxic" goiter may exist as an entity caused by the action of a modulator of thyroid hormone activity.

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CURRENT REAPPRAISAL OF TOTAL ABDOMINAL HYSTERECTOMY*

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SINCE the advent of meticulous preoperative preparation, which includes careful systemic inventory, the medical management of deviations from normal, the restoration of blood loss and the use of antibiotic chemotherapeutic agents the patient becomes a better candidate for gynecologic surgery. With the improvement of anesthesia and more cooperation between the surgeon and the anesthetist, with the use of longer incisions with resultant better surgical exposure, the emphasis of importance of the gentle handling of tissues and the administration of fluids, blood or plasma during surgery the patient is brought to her immediate recovery phase better than at any time heretofore. Moreover, advances in the knowledge of electrolyte balance, the giving of blood, plasma or fluids postoperatively, the intravenous administration of proteins and vitamins, early ambulation and the use of sulfa, penicillin or streptomycin all contribute for a speedier return to normalcy.

Today our fellow general surgeons can perform various extensive major surgical operations which often take five or six hours and may require four or five transfusions during surgery. Esophagectomy with supra-aortic intrathoracic esophagogastric anastamosis, total collectomy, pneumenectomy, total gastrectomy or abdominal perineal resection are done with comparatively low mortality rates.

It is a combination of all the aforementioned factors which have again swung the pendulum toward total abdominal hysterectomy. Total hysterectomy is more advantageous than subtotal hysterectomy because of the possible occurrence in the latter of carcinoma in the cervical stump; and the discharges due to endocervicitis, erosion, eversion or to circulatory changes subsequent to supracervical hysterectomy. It is also advantageous because of the absence of troublesome bleedings from the cervical stump which may be due to inflammatory disease due to cervical polyps, hypertrophy of the endocervix or the remnants of a small portion of endometrium. In a rare case strictures of the cervix may produce a hemato-or pyocervix.

Some observers are of the opinion that trichomonads or fungi may lodge in the crevices of the endocervix and be a site in the reinfection of these troublesome leukorrheas. With the emphasis on a "back to fundamentals" movement, the gynecologic picture interpreted in anatomic, physiologic, and associated pathologic thinking has markedly decreased the feared "Big Two" damage in total hysterectomy, namely, the ureters and the bladder.

We will attempt to show this in our discussion of the simplified technic used in total hysterectomy.

ESSENTIAL SURGICAL ANATOMY

The ureters in their entire transpelvic course are retroperitoneal. In the region of the adnexa the ovaries make a depression in the peritoneum called the fossa ovarii with the ureter as the posterior boundary. It is at this point that the ureters are not infrequently damaged while the adnexal portions are being removed. To avoid such possibilities the lateral masses must be elevated and separated from the posterior

peritoneum and then, and only then, clamped and divided. Application of curved clamps to fixed lateral adnexal organs without preliminary separation may lead to ureteral damage. Ureters are damaged more frequently at this site than they are further down along the uterus and cervix.

As the ureter courses downward the uterine artery crosses over it at the lowest portion of the broad ligament. At this point it lies lateral to the cervix approximately a finger-breadth, or about 1.5 cm. away, where it courses further down and enters the urinary bladder posteriorly and obliquely.

The cardinal ligaments (Mackenrodt's ligaments) which are actually the lowest portions of the broad ligaments are the chief supports of the cervix and upper vagina. They are composed of connective tissue, muscle fibers and massive venous plexuses. These ligaments often give rise to a great deal of venous oozing if they are not properly clamped and transfixed. But caution must be exercised to avoid injury to the ureters while clamping the above ligaments. We always stay close to the cervix when applying the clamps and always converge the tip of the clamp toward the cervix proper. The transfixion needle should not take too much tissue in its bite. We frequently use the word "hug" the cervix to imply the closeness of the application of the clamps.

The cardinal ligaments continue posteriorly together with the uterosacral ligaments which form the chief support of the uterus and the cervix posteriorly. The latter must be dissected to liberate the cervix and the vagina for complete removal of the uterus. The urinary bladder is attached to the corpus uteri by fascial vesico-uterine ligaments and to the vagina by the vesicocervical ligament.

The peritoneal vesical reflexion on the uterus and the fascial connections must be pushed or rolled down medially to avoid bladder injury and pushed down laterally to reflect the ureters further away from the field of operation to avoid their damage.

One can feel with the examining fingers the ureters at this point as firm cord structures. When the bladder and its fascial attachments have been reflected down past the cervix, the latter can be palpated easily and at this time the vagina is the only structure which is attached to the cervix.

TECHNIC

The patient is prepared for surgery in the manner suggested in our opening statement. At the time of operation either fluids or blood are administered intravenously. It is advisable and convenient to have the fluids running intravenously in the ankle but if the arm is used, use only the back of the wrist or the cubital fossa of the forearm. The arm must be next to and parallel with the patient. We have been using the Wells arm protector which has been very efficacious. Prior to its use we have seen two patients with temporary damage to the nerves in the brachial plexus with resultant transient paresis of the arm. This was due to hyperextension and extreme abduction of the arm by the inadvertent shifting of the surgeon or the assistant.

The patient is placed in a deep Trendelenburg position and if adequate surgical exposure can be obtained by an infraumbilical incision, it is used. The incision must start on the skin covering the bony symphysis and not 1 or 2 inches above the symphysis at the hair line. In other cases, and there are many, it is necessary to extend the incision to the left of the umbilicus. The left side is used rather than the right side of the umbilicus to avoid the round ligament of the liver. If a patient has had a previous midline incision, we always excise the old scar and then extend the superior skin incision 1 or 2 inches longer. The peritoneal cavity is always opened above the linea semicircularis, and our preference is the superior angle of the incision. In a patient who has not been operated upon before we may avoid entering the urinary bladder, which may be congenitally high or be raised by a pelvic inflammatory process or an endometrio-

sis, neoplasms or a malignancy. We believe that it is very important to emphasize the fact that in a reoperative case the peritoneal cavity should be entered in the superior angle of the incision just below the point where the old skin excision was extended, thereby reducing the chance of bowel, bladder or omental injury. Additional points which will benefit the surgical exposure are: to incise the fascia down to and touching the symphysis pubis; not to stop in the inferior angle opening of the peritoneum when one ceases to see the translucency of light, but to feel for and make the incision of the peritoneum almost to the dome of the bladder.

The pelvis is cleared of bowels with warm lap pads in a roll-away fashion and then the corpus uteri is grasped with a tenaculum. In cases in which only the uterus is to be removed two straight hemostats are placed on the right and then on the left broad ligament in close proximity to each other pointing toward the cervix, with the inner one touching the uterus. These include the tube and the utero-ovarian ligament. An incision is made between them and the outer forceps is transfixed. The next two forceps are applied inferiorly and include the round ligament; the latter is divided and the outside one is then transfixed. This same procedure is continued on one side down to the corporocervical junction, and then the same procedure is accomplished on the opposite side. At this stage of the operation it is often possible to take off all of the forceps which were attached to the uterus for the purpose of controlling the backflow and keeping the field dry. A transverse incision is now made in the peritoneum just above the bladder. This is often simplified by picking up the bladder with a toothless tissue forceps and then making the incision in the peritoneum which reflects on the uterus. Occasionally, when dealing with an inflammatory process or an endometriosis (or even in an uncomplicated case), if one exerts rhythmical pressure downward, pushes the cervix down into the vagina and then exerts trac-

tion upward with slight extension on the peritoneal reflection, it will often simplify finding the exact attachment of the peritoneum and prevent potential urinary bladder injury. With the back end of a long tissue forceps, a cleavage plane is then found between the cervix and the uteropubic fascia, and this is reflected off the cervix and vagina together with the bladder. The direction of this reflection should be bilateral as well as inferior and in that way the ureters will be pushed to either side. A posterior transverse incision is then made in the peritoneum superior to the attachment of the uterosacral ligaments. We believe that the more peritoneum that is available posteriorly the less subsequent rigidity of the floor of the bladder and thereby less postoperative bladder symptoms which are occasionally seen after a total hysterectomy. The posterior peritoneum and uterosacral ligaments are then separated from the cervix and vagina. This high incision in the posterior peritoneum will also protect against possible injury to the rectum. The paracervical tissue or cardinal ligaments are then clamped with single forceps close to and hugging the cervix. This hugging of the cervix with the tip of the forceps pointing medially (converging) is another paramount factor in preventing ureteral injury. The above are then incised and transfixed down to the vagina first on one side and then on the other.

At this stage of the operation the only structure which is holding the uterus in the abdomen is the vagina. There are no clamps attached to any structures in the pelvis at this time thereby giving us a clear field to work in. Next two Boui rectal clamps are placed underneath the cervix on either side, an incision made above them and the entire uterus lifted out of the abdomen. The clamped vagina is now sewed with an over-and-over chromic No. 1 suture, and by rotation of the Boui clamps away from the bladder the vagina can easily be identified and the bladder not incorporated in this suture. The vaginal
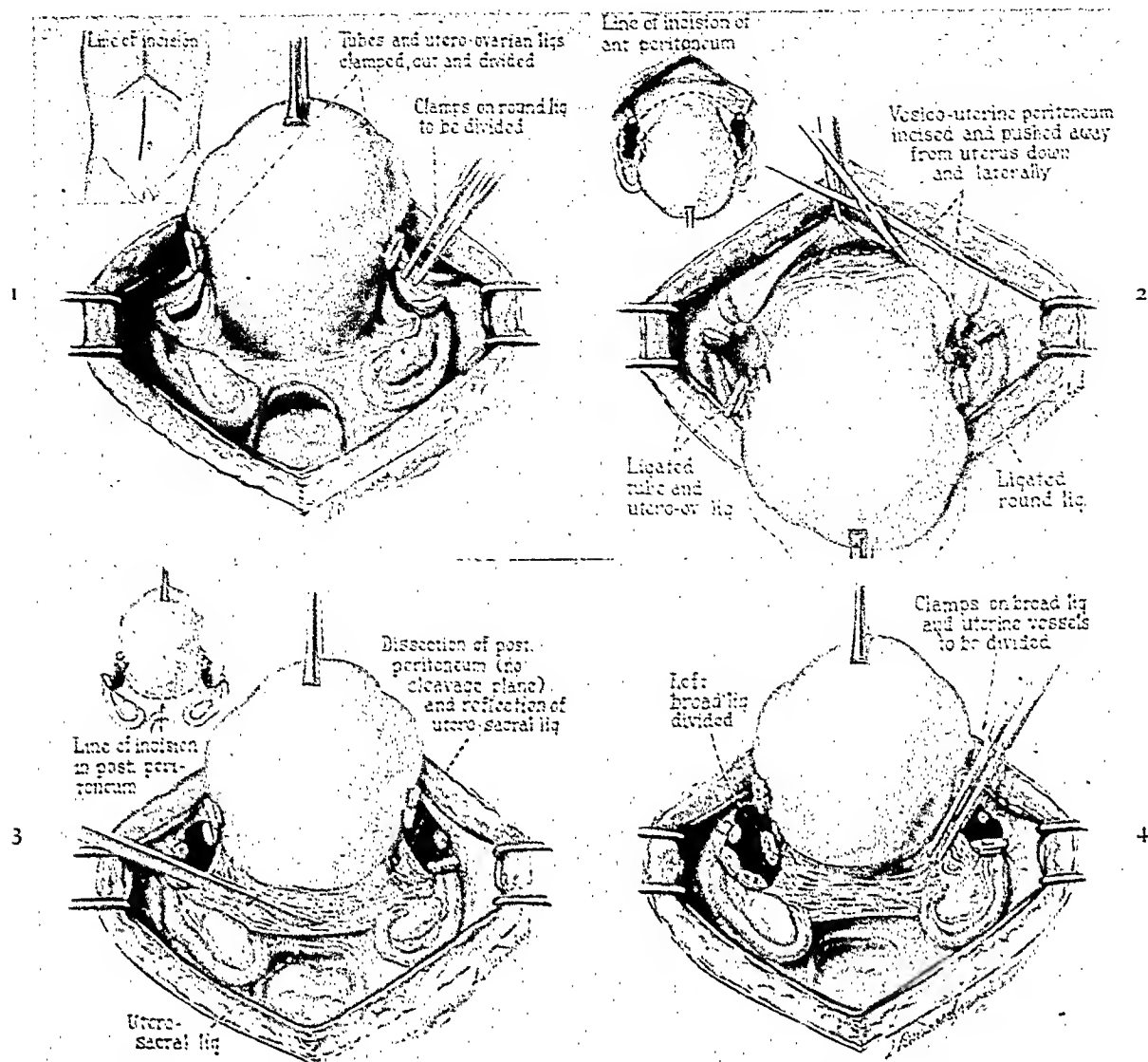
FIG. 1. Clamping and dividing of tubes, utero-ovarian and round ligaments.
FIG. 2. Reflection of vesico-uterine peritoneum downward and laterally.
FIG. 3. Dissection of posterior peritoneum and reflection of uterosacral ligaments.
FIG. 4. Division of the broad ligaments.

vault is supported by a silk suture posteriorly and anteriorly, to include the uterosacral ligament, the angle of the vagina and the cardinal ligament on either side. The round ligament is then sewed to the vagina on either side with a heavy silk suture, and the posterior peritoneum is sewed to the anterior peritoneum with interrupted fine silk sutures. The excess posterior peritoneum makes this subsequent peritonization free and non-rigid which, as we mentioned previously, is probably a factor in decreasing the number of patients who have bladder symptoms subsequent to hysterectomy. Two interrupted medium silk sutures are placed

transversely into the uterosacral ligaments. The purpose is first, to prevent a potential enterocele which can be the etiology for a subsequent vault prolapse; second, it also has a tendency to keep the vagina more forward and third, it may be an additional factor in preventing the bladder symptoms of urgency and terminal dysuria.

If the adnexae are to be removed in addition to the uterus, we prefer to do the salpingo-oophorectomy prior to the start of the hysterectomy and transfix all clamps to give us a clear field to work in. However, in the case of pelvic inflammatory disease or endometriosis, it is often necessary to remove the uterus first thus giving the sur-
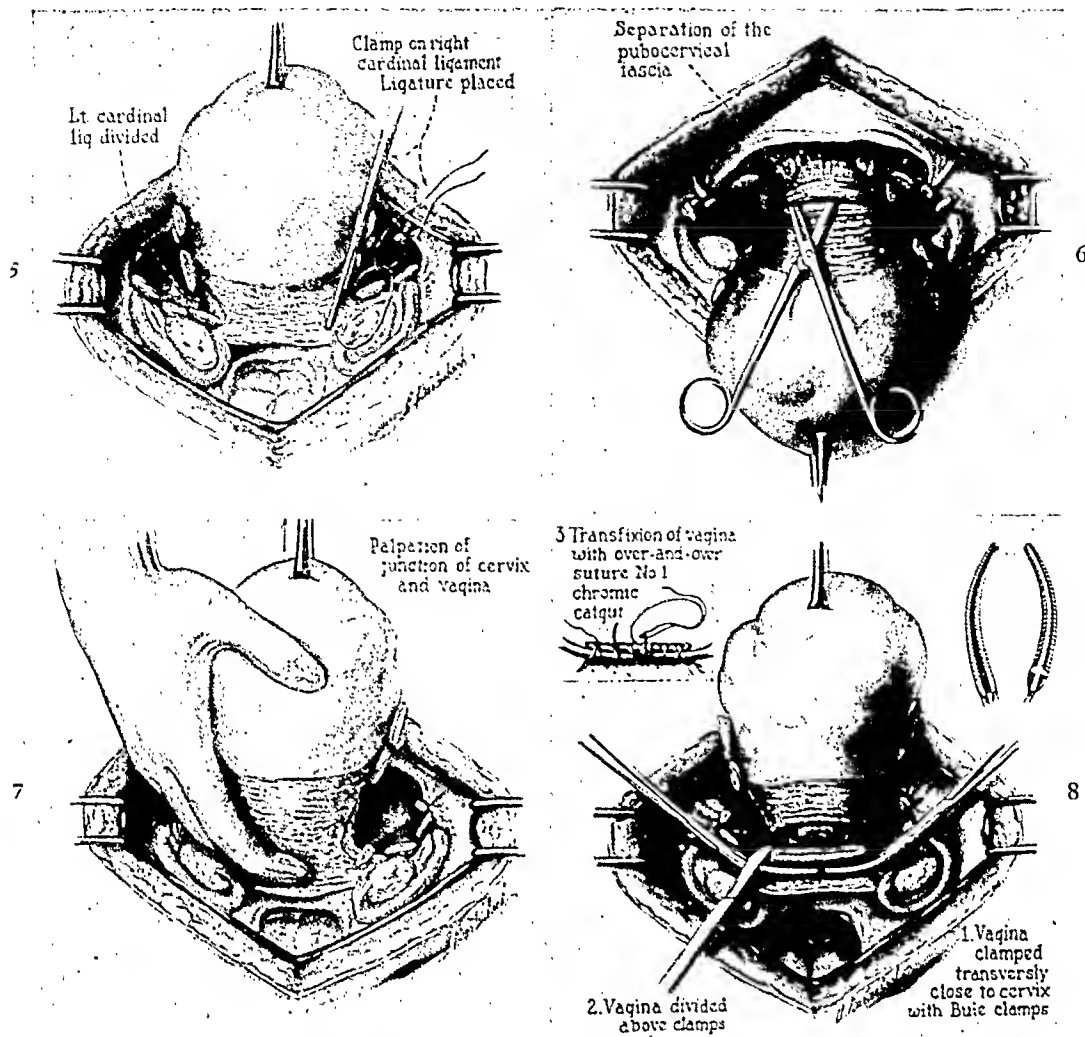
FIG. 5. Clamping of cardinal ligament.
FIG. 6. Separation of pubocervical fascia.
FIG. 7. Palpation of junction of cervix and vagina.
FIG. 8. Vagina clamped transversely with Buic clamps.

geon ample room and better vision to remove the adnexae with the accompanying adhesions. (Figs. 1 to 12.)

COMMENTS

We believe that with proper training of the surgeon complete removal of the uterus will eventually replace the subtotal operation. Not infrequently the question is raised regarding the depth of the vaginal vault after total hysterectomy. Is it shortened? What about dyspareunia, vaginal vault prolapse, the physiologic appearance of the vaginal mucosa, libido, urinary bladder dysfunctions, and mortality or morbidity as compared with the subtotal hysterectomy? These pertinent questions were answered as we followed many cases of total hysterectomies. The vaginal vault was not shortened if it was cut close to the cervix when the uterus was removed. Dyspareunia is another query of many doctors but here, too, in our series we did not see such a complication following the aforementioned surgical procedure.

We previously saw several patients with prolapse of the vaginal vault following complete hysterectomy. In the previously described method of vaginal vault support we have not as yet seen a prolapse in the cases we have been able to follow. Why the prolapse occurred may be difficult to
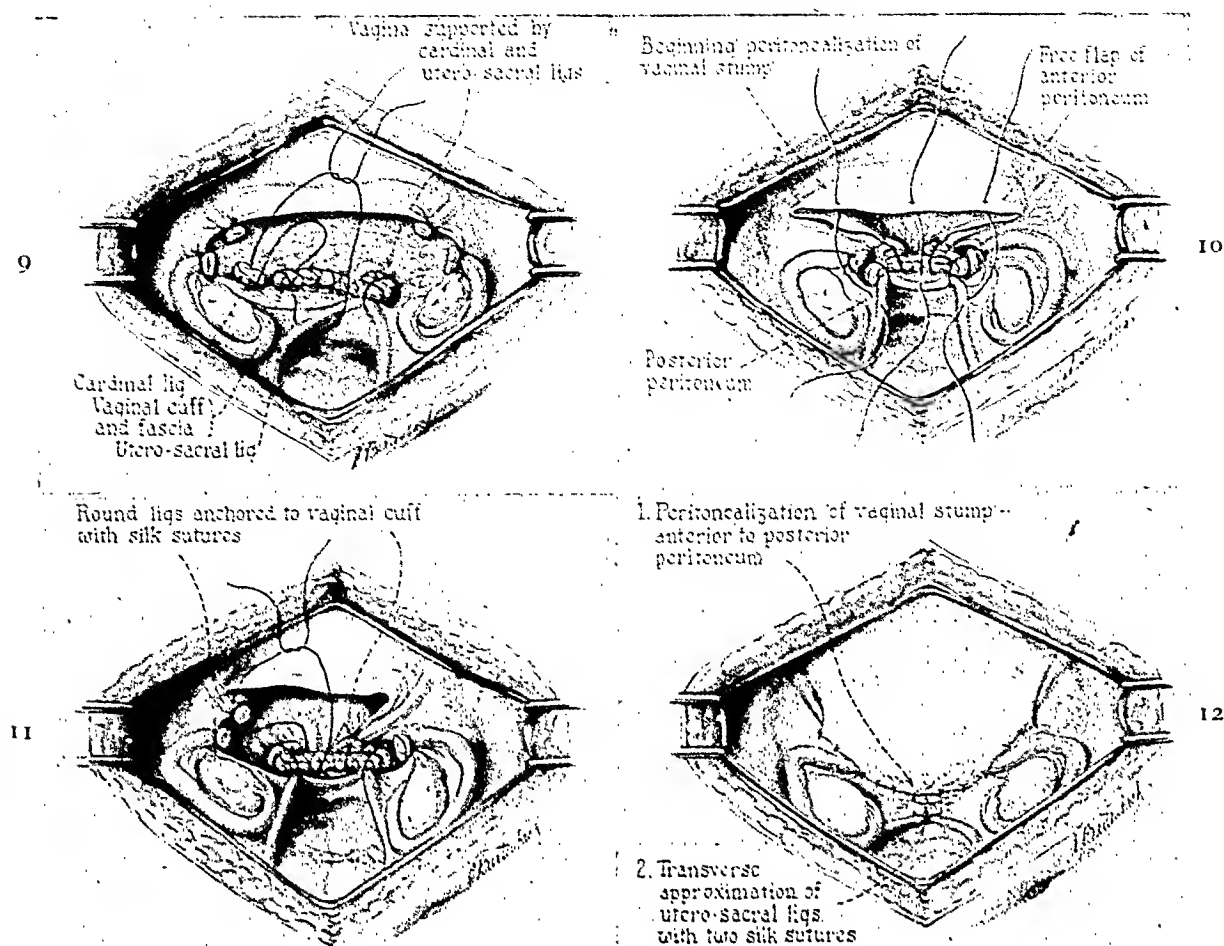
FIG. 9. Vagina supported by cardinal and uterosacral ligaments.
FIG. 10. Peritonealization of vaginal stump.
FIG. 11. Anchoring of round ligaments to vagina.
FIG. 12. Peritonealization completed; transverse approximation of uterosacral ligaments.

answer, but improper support of the vagina, poor inherent tissue tones or defective sutures, either due to a low grade infection or loss of tensile strength, may account for such a prolapse. Any one or a combination of the aforementioned factors may be responsible. We employ silk for vaginal vault support.

The vaginal mucosa alone following a total hysterectomy is not affected. It appears succulent, thick and moist. Even patients in whom the ovaries are removed at the same time fail to show any immediate evidence of atrophy, immediate meaning within one year.

Libido is a variable reaction among patients. Many describe a definite improvement which must be psychic, in that there is ease of mind in regard to conception; others are *status quo* as prior to surgery

while a small percentage report some decline.

We have been aware for a long time that urinary bladder complaints following hysterectomies were a bothersome problem. Because of this, the urinary bladder proper and the peritoneal reflection of the bladder receive special handling and consideration. By pushing the fundus of the uterus and the cervix with the attached tenaculum down and up several times one can detect the bladder and peritoneal reflection in the uterus very easily. This establishes the exact urinary bladder peritoneal attachment on the uterus. The line of incision, therefore, is made high on the uterus, giving a free and a large peritoneal flap for future free peritonealization. This step prevents what is so frequently done, namely, to sew the bladder flap under too

much tension to the posterior peritoneum which is fixed, giving rise to elongation and rigidity to the bladder. The latter will produce symptoms of urgency and frequency of urination in the absence of urinary infections and without any organic disturbance. The transverse suturing of the uterosacral ligaments is also probably a contributing factor by holding the vagina forward.

Morbidity and mortality rates in our series are essentially about the same as for those undergoing subtotal hysterectomy. Early ambulation is routine in these cases. To minimize morbidity we routinely use in all cases of total hysterectomies 50,000 units of penicillin as the initial dose and 25,000 units every three hours for three or four days postoperatively. This may be and probably is an important factor to reduce the frequency of infection and indirectly will reduce the frequency of postoperative vaginal hemorrhage which may be due to sloughing of a suture as a result of a low grade infection.

As an additional and routine prophylactic procedure against possible infection the vagina is "surgically prepped" in the same fashion as the abdomen, namely, a soap and water scrub for a period of ten minutes with frequent flushing with water, then an alcohol rinse and finally it is painted any one of the popular antiseptics. We do not use iodine.

Use of gelatin or other absorbable styptic sponges over raw or oozing surfaces undoubtedly is also valuable in preventing hemorrhages.

SUMMARY AND CONCLUSIONS

1. An appeal for total hysterectomy is made. It is not new but a current discussion of the problem.

2. A discussion of pre- and postoperative measures is presented.

3. Reasons for complete removal of the uterus and cervix are discussed.

4. The technic of the operations is herewith outlined.

5. A discussion is presented of the various practical and common questions which are often raised by fellow colleagues.

REPAIR OF FASCIAL DEFECTS WITH WHOLE SKIN GRAFTS

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THE presence of fascial defects has presented a difficult problem to the surgeon. Various methods of repair of these fascial defects have left much to be desired. In the past attempts to eliminate these defects consisted of the use of a fascial graft or fascial strips. It has been found difficult to obtain either pedicle or free fascial grafts of homogenous fascia in sufficient quantity to obliterate large defects without creating a new defect or at least a weakening of the donor area. Probably the method which has been most applicable is the use of fascial strips as a lacing suture or weaving material similar to the mending of a hole in a sock.

Lack of abundance of fascial tissue and the difficulty of procurement of the grafts suggested a search elsewhere for a tissue that might be substituted for fascial tissue. It was believed that the substitute tissue should possess several characteristics peculiar to fascia and in addition be more readily obtainable, stronger, if possible, and more amenable to transplantation. The new tissue should have a good blood supply in contradistinction to the avascularity of fascia in order that a "take" would be insured in a high percentage of cases. It is not difficult to visualize, through elimination, that the only tissue that had most of the desired characteristics was the skin. This skin had been successfully grafted in the past to replace other skin and in deeper tissue to fill depressions. The dermal, fat and fascia grafts have been a part of the plastic surgeon's armamentarium when their employment has eliminated deep tissue defects and superficial deformities. As long as the asepsis was not violated these grafts took well. The super-

ficial layer of skin was removed. This usually involved all of the stratum cornium and a most superficial portion of the stratum granulosum. This is a split-thickness graft of approximately 16/1,000 of an inch.

This type of graft (dermal-fat-fascia) was employed in one case in which there was a large soft tissue defect on the lateral aspect of the right thigh. The defect in the fascia lata was ovoid in shape measuring 15 by 10 cm. It was decided that a dermal-fat-fascia graft would best reconstruct and repair the defect. The defect was so large that it was believed to be necessary to raise two dermal-fat-fascia grafts from the anterior abdominal wall. These two grafts were sewn together with their synonymous layers in juxtaposition. The fascial layer was sutured to the freshened edges of the fascia lata defect. Recovery was uneventful until the twenty-third postoperative day. At this time there appeared an area of fluctuation in the region of the distal portion of the graft. An incision was made and it was found that the fascia was not healing to the surrounding tissue. The fascia was also separating from the fat of the original graft. The serum and fluid present suggested that the fascia and, or the fascia and the cotton sutures used in fixation was acting as a foreign body. The fascia was removed in the hope that the rest of the graft might be saved as both the fatty and dermal portions seemed to be viable. Three months later the fascial defect had become obliterated completely and the muscle no longer herniated through this region. However, there was a soft tissue defect residual with fixation of the skin to the subcutaneous scar tissue. The fixation was annoying

and the patient requested that the existing defect be corrected. Another dermal-fat-fascia graft was prepared. When the skin of the thigh had been dissected free of the subcutaneous scar tissue, there was found a definite layer of tissue firmly attached to the fascia lata that could have been no other than the dermal layer of the graft. It had made a firm and solid closure of the old fascia lata defect which had a resistance similar to the surrounding fascia. The more superficial defect was obliterated with the second graft which was made up of dermal and fat layers without fascia. This took successfully and the defect was completely alleviated.

This chance experience had opened a new vista and it had suggested a new tissue to be substituted for fascia in the repair of fascial defects. Apparently there was no need for the inclusion of the fascial layer in the dermal-fat-fascia graft. In fact, the inclusion of the fascia narrows the field open as the donor site and unnecessarily complicates the technical procedure. The dermal-fat graft or the dermal graft offers an ideal tissue which is easily obtainable in large quantities, easy to handle, about as strong as fascia and freer from complications.

The technic of obtaining the dermal graft more or less depends upon the existing defect that is to be repaired. However, there are certain general procedures that are applicable to the technic of obtaining any of the grafts. The preoperative preparation of the donor skin must be meticulous. The area should have a forty-eight-hour preparation with the following technic. The area is carefully shaved and scrubbed with green soap, water and a brush for a period of ten minutes. The soapsuds are then rinsed off with warm sterile water. Seventy per cent alcohol is applied. This is followed with ether. The area is then draped with a sterile towel which is held in place with a bandage. It has been found that mercurial antiseptics should not be employed in the preparation of skin that is to be used for grafting. The above procedure is repeated the next day and again at the operating table. After the patient has been draped and the recipient site has been prepared the initial 15/16/1,000 of an inch of the superficial skin, which includes the stratum cornium and a portion of the stratum granulosum, is removed by a Blair knife with attachment or a Paggett dermatome. The author prefers the use of the dermatome. The remainder of the dermus and the desired subdermal tissue is then excised according to the required pattern. The linear shrinkage of approximately 20 to 25 per cent must be taken into consideration when the pattern is laid out. The depth shrinkage is much greater than the linear shrinkage. It may be as great as 50 to 60 per cent. However, the latter type of shrinkage has a retarded nature as it is discernable only after two to three weeks after the placement of the graft. This shrinkage is directly proportional to the amount of fat that has been included with the graft. The defect in the donor area is closed by undercutting of the edges and by the use of retention sutures. It has been found that the deep Halsted type of retention sutures are most satisfactory. If the graft has been a large one, it may be necessary to roll the subcutaneous tissue into the defect after the undercutting has been carried deeper and toward the deep fascia. If the skin is under much tension, a complete closure with the retention sutures should not be attempted. After a partial closure has been made with the retention sutures the remainder of the skin defect may be covered with the split-thickness graft that was originally removed before the dermal-fat graft was raised. The author has employed cotton sutures throughout, No. 100 for bleeders and non-tension suturing and No. 36 for retention sutures. All surfaces are anointed with a thrombin solution. The split-thickness grafts are retained with Zeno adhesive or a modification thereof. They are never sutured. The use of any adhesive of this type necessitates a completely dry field. If the split-thickness graft has been permitted

to remain on the drum, there is no need for the use of saline and the graft will be dry when it is applied. A light coat of warmed sulfathiazole ointment 5 per cent is brushed over the graft or the area of closure. A sterile gauze dressing is applied and covered with sterile cellophane or wax paper. Fluffed gauze or mechanics waste is bandaged or strapped in place for pressure. This dressing is left *in situ* from ten days to two weeks unless the odor or a drainage suggests a catastrophe.

The technic of application of the dermal or dermal fat graft depends upon the requirements of the defect to be repaired. In all cases thrombin solution is used to anoint all of the contacting surfaces. The dermal edges are sutured to the freshened edges of the fascial defect. No attempt was ever made to fix the fat. A pressure dressing similar to that described above is employed in each of the ensuing cases.

Muscular herniations through the fascial defects of the extremities present a problem that pure fascial grafts do not successfully answer. Naturally smaller defects in the deep fascia, such as those occasionally seen lateral to the anterior crest of the tibia, may be caused when plicating the fascia with sutures. However, there may be a great deal of tension created which is unnecessary. There is usually a loss of subcutaneous tissue undoubtedly due to the pressure of the muscle that has herniated through the fascial defect. If the rupture is small, the superficial defect is readily obliterated by sliding the edges together with undercutting of the edges and retention sutures. However, if the defect is a large one, it is preferable to introduce the dermal-fat graft placed upside down.

This method in various ramifications has been employed in seventeen cases of muscular herniation in the extremities and other soft tissue defects of the extremities involving the deep fascia. Three of these cases were apparently congenital fascial defects as it was impossible to obtain a history of trauma either new or old. The remaining fourteen cases were definitely post-traumatic fascial defects (lacerations, gunshot wounds, etc.) with and without hernia.

CASE REPORTS

CASE I. W. S., age thirty-seven, sustained a gunshot wound of the left leg about 6 inches below the knee in November, 1942 that was débrided within two hours of the injury. Five days later a secondary closure was effected and the limb was immobilized in a long boot, circular splint of plaster of paris. After two weeks passed the wound was redressed through a window cut in the splint. The healing seemed to be progressing satisfactorily. After the termination of the eighth week when the splint was finally removed and the patient was permitted to stand without support, much to the surgeon's surprise there was a herniation of the fascia just lateral to the tibial crest about the size of a fifty cent piece. The decision to repair the herniation was made only because the patient complained of the disfigurement created by this herniation. There was no associated symptomatology. The dermal-fat graft was decided upon and the graft was obtained from the abdomen and it was placed fat-side externally over the defect. The dermal portion denuded down to and including the superficial portion of the stratum granulosum was sutured to the freshened edge of the fascial defect. The skin was closed and a mechanics waste pressure bandage supported by a light plaster boot was applied. The leg was redressed after the plaster boot had been bivalved on the tenth day. The healing seemed to be progressing satisfactorily. The bivalved plaster was replaced for support for the next three weeks. The plaster was removed daily for the purpose of giving massage and regulated active exercises. Warm whirlpool baths were instituted after the second week. After two months the defect was left unsupported and there was no suggestion of the herniation present.

CASE II. E. McC., age twenty-two, who received a shell fragment wound of the lateral aspect of the right lower leg mid-portion on June, 1944, represents a slightly different treatment of the soft tissue with fascial defect with the dermal-fat graft. As there was a large defect in the muscle in addition to the fascial defect a double dermal-fat graft was decided upon. Initial débridement of the wound with

removal of the foreign body had been effected less than an hour after the injury had been incurred. The patient came under the care of the author on the tenth post-traumatic day without any attempt at secondary closure. After the third day of penicillin lavage the granulations appeared contamination-free and the reparative operation was performed. The two dermal-fat grafts were obtained from the anterior abdominal wall. They were deprived of the superficial layers. The two dermal layers were placed in a juxtaposition with one pad of fat filling the muscular defect and the other making up the lost subcutaneous tissue. The whole area was then covered with the split-skin graft removed from the donor area initially. In this case thrombin and leukocytic adhesive was used after the formula of Dr. M. Zeno in the hope that the introduction of new blood supply would be enhanced where the multiple grafts were concerned. The two dermal-fat grafts took well but the center of the split-skin graft sloughed off. This slough was not surprising for the graft had been applied as a dressing for the area denuded of superficial skin. After the first eight days had passed there was a good base of granulation tissue in this area. After two days of continuous penicillin jelly (penicillin 500 units in quinolor jelly per 1 cc.) application with a pressure dressing, the granulations were pink and healthy looking. A second split-thickness skin graft was transplanted and this graft took without difficulty.

The case mentioned earlier in this paper of the large muscular herniation through the fascia lata is an example of the use of a double graft. However, the two grafts were not transplanted simultaneously. The other cases of this series are of sufficient simularity that it seems a definite waste of space to consider each of the cases singly.

The facility with which the dermal-fat grafts may be handled is surprising to the surgeon who has not employed this type of graft. The percentage of successful takes is high if absolute asepsis is maintained and the graft is carefully sutured in place.

Fascial defects of the abdominal wall in the thirty cases that have been repaired with the use of the dermal-fat graft fall into two groups; the repair of hernias and the initial repair of fresh defects.

Venteral hernia which is most frequently a postoperative or incisional hernia presents a large defect in the fascia which must be closed by the approximation under tension of the more or less attenuated fascial edges or closed by the introduction of new or transplanted tissue. If the defect is very large, it is nearly impossible to effect a solid and satisfactory closure with the introduction of a graft of tissue. With a graft, the tension factor can be eliminated and the end result lends a more insured success.

CASE III. Mrs. E. L., age fifty-eight had had a laparotomy performed in 1932. She was found to have a large incisional hernia the size of a grapefruit in the lower right rectus region. The original incision apparently had been a lower abdominal right rectus incision. The abdominal skin over the hernia was laid back by a crescent incision over the median portion of the region of the protruding mass (the defect) and dissected free of the fat and scar tissue. The edges of the superficial rectus sheath were dissected free. The existing fibers of the attenuated rectus muscle were isolated from the fascia and scar tissue. The deep or posterior portion of the rectus sheath was separated as much as possible and the scar was excised with a small portion of the attached peritoneum. The peritoneum was then closed but it was impossible to create an approximation of the posterior rectus sheath without the sutures tearing out of the tissue due to the excessive tension required to effect the closure. The plan of operation had included the repair of the defect in the anterior rectus sheath with a dermal-fat graft, but it was not believed that it was possible to obtain an additional graft without difficulty. Strips of skin from the edges of the surgical incision were removed. The outer layers of the dermus were removed and the remainder of the strips were woven together to close the defect. The rectus muscle was replaced into its proper position and sutured lightly to the repaired area. A large dermal graft was prepared and raised. It was then placed into the defect in the superficial rectus sheath with the fat side upward and sutured to the edges of the sheath. The skin

was closed over the graft by sliding the edges together.

CASE IV. Mr. H. K., age forty-four, presented himself September, 1940, two years after recovering from a posterior gastroenterostomy with a hernia about 6 cm. in diameter in the upper abdominal midline incisional scar. In view of the fact that this patient had little or no subcutaneous tissue, a dermal graft without fat was employed. This graft was sutured without tension into the defect in the interrectus apponeurosis. The herniation did not recur and after three months it was impossible to discover any suggestion of the old fascial defect.

Similar methods have been employed to repair defects in upper right quadrant incisions for cholecystectomy. The two following cases are among the cases of this series which were of a severe nature.

CASE V. Mrs. B. G., age fifty-four, sustained an infected biliary fistula which followed an abscess that caused damage to a large portion of the operative incision which was of the oblique type following the costal margin. After seventy-eight days of irrigation had been prescribed with the adjunction of oral sulfa chemotherapy it was found necessary to excise a portion of the tenth rib which exhibited osteomyelitic changes on x-ray. Following operation, the drainage became minimized and it was believed that the remaining fistulous tract could be removed and the incision repaired. As a large portion of tissue had been removed and there was a wide separation of the incision borders the use of grafts was found mandatory. On the ninety-sixth original postoperative day the fistula and a large portion of scar tissue were excised. The peritoneum was closed with the aid of transplantation of the falciform ligament. A dermal-fat graft with only a thin layer of fat was sutured in place in the defect of the submuscular fascial tissue with the fat in juxtaposition to the peritoneum. The musculature was drawn together loosely. Another dermal graft without fat was used to complete the deep fascia. Both grafts were sutured to the periosteum and perichondrium of the tenth rib. The abdominal subcutaneous tissues and the skin were closed by a type of Szymanowski plastic flap transplantation.

CASE VI. Mrs. E. E., age forty-one, had a large incisional hernia as a sequel to a cholecys-

tectomy which had been performed in May, 1942. In December, 1943, the hernia was repaired. Again the falciform ligament was employed to eliminate in the peritoneum any tension that would have occurred if an attempt had been made at simple closure. A strip of skin with the superficial layers denuded was used to fortify the peritoneum. The muscle was closed by splitting and sliding together in the form of pedicles. A dermal graft was sutured in the deep fascial defect. The skin and subcutaneous tissue were closed by undermining the edges and the use of retention sutures.

The dermal or dermal-fat grafts have been employed to repair initial abdominal wall defects with fascial defects.

CASE VII. Mr. H. C., age fifty-six, complained of pain in the upper left abdominal quadrant. He gave a history of attacks of indigestion for the prior seven to eight months. He had lost 68 pounds during this period. There was a mass about 5.5 cm. in diameter that was palpable in the left upper abdominal quadrant just lateral to the lateral border of the left rectus muscle. This mass seemed fixed to the abdominal wall. A barium enema revealed a defect in the transverse colon in this region. At operation it was found that there was a large tumor apparently arising from the wall of the transverse colon at the attachment of the mesocolon. This mass was also attached to the abdominal wall anteriorly where it seemed to be growing by contiguity. An anastomosis was completed in the transverse colon following removal of the tumor. It was necessary to excise a large portion of the anterior abdominal wall (about 7 cm. in diameter) in order to remove the tumor and the adjacent tissue. This procedure was closed with a free graft of omentum. The apponeurosis of the oblique muscles was repaired by a dermal graft. The skin was closed without difficulty as none had had to be sacrificed. The tumor proved to be a sarcoma. The patient was given a course of x-ray radiation. It is interesting to note that this therapy had no deleterious effect upon the graft so far as could be determined clinically.

Several inguinal hernias have been repaired with the aid of strips of skin used to fortify the fascia or by actual dermal grafts.

It is believed that an unusually strong repair may be effected in the direct inguinal hernias when the conversion of the direct hernia to an indirect hernia has been effected and the floor of the canal fortified by a graft of dermal tissue. The remainder of the repair may be completed by any of the accepted procedures.

The other cases of this series offer no other features that would make a recital of their case histories essential. Introduction of these histories would only progress toward a more definite realization that this method of fascia repair by dermal or dermal-fat graft is simple, certain and dependable. The technic of raising the graft and its transplantation is not frought with elaborate nor time-consuming methods.

CONCLUSIONS

1. The dermal or dermal-fat graft is an ideal tissue with which to repair fascial defects because it is obtainable in comparatively large quantities, easy to handle, about as strong as fascia grafts and less susceptible to complications.

2. These grafts may be employed to repair incisional hernias, inguinal hernias, muscular herniations of the extremities and initial fascial defects.

RADLOFF and King studied fifty cases of esophageal hiatus hernia and found that other diseases were also present in two-thirds of the cases (66 per cent) and that, in most instances the patients' symptoms were due to this concomittant disease. Surgery was needed in only three cases; undoubtedly, most of a patient's stomach may be up in her chest and still the patient never has any symptoms. I, personally, have encountered several such instances in which routine fluoroscopic examination has shown the existence of such a hiatal hernia without symptoms. The operation for repairing these diaphragms is not a simple one and should not be done unless urgent clinical symptoms are present. (*Richard A. Leonardo, M.D.*)

SIMPLIFIED TECHNIC FOR SUBTOTAL THYROIDECTOMY*

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REMOVAL of the diseased thyroid gland has been a challenge to the technical skill of surgeons for many years. The extreme vascularity of the gland, its proximity to the recurrent laryngeal nerves, and the intimate relation with the trachea, parathyroids and carotid sheaths have magnified the technical difficulties attendant to either partial or complete extirpation of the gland. All surgeons who see large numbers of thyroid abnormalities have devised various methods and have many technical details of their own. That numerous variations in technic still exist is not to be denied for every surgeon puts into his work the seal of his own individuality; consequently, divergence of details will always be found.

Only recently have there been a number of articles on the technical aspects of thyroid surgery from the large clinics over the country. We are proposing a simplified procedure, sound in principle and efficient in execution, the technic of subtotal thyroidectomy evolved by the senior author (U. M.) and used routinely on the Louisiana State University Surgical Division of Charity Hospital. In addition to other technical variations emphasis is placed on early division of the thyroid isthmus *en masse*, using specially designed isthmus clamps (Kuker) before directly attacking either lobe of the thyroid gland. A portion of the paper and illustrations will be given over to pertinent details which led to the design of the isthmus clamp, a description of the instrument and the rationale of its use in thyroid surgery.

On numerous occasions in the past it had been observed that division of the thyroid isthmus was very difficult with the available instruments; this was especially so in obese patients. Temporary respiratory embarrassment and tearing of the gland and contiguous structures, with consequent hemorrhage, were frequent complicating and occasionally lethal factors. Technical skill is in no small measure dependent on the surgeon's proper choice and use of instruments. In the past there was no recourse to an instrument which adapted itself to the maneuver of dividing the thyroid isthmus.

CLINICAL STUDIES

It is obvious from clinical inspection and anatomic measurements that the trachea forms a definite but varying angle with the anterior chest wall and especially with the manubrium sterni. To verify this angulation actual measurements were made on one hundred different patients. This procedure consisted of placing one limb of a modified protractor on the flat surface of the manubrium sterni and the other limb parallel to the anterior aspect of the neck with the patients in a relaxed supine position, using the suprasternal notch as the center point of angulation. This series of patients comprised a rough cross section of the various age groups and varied in build from thin to obese, both white and colored. There was a wide variance in the angulation, ranging from 25 degrees in the thin, flat chested patients to 65 degrees in the robust, obese patients, the mean average was 40.3 degrees. From these findings it is apparent that a straight instrument applied to the thyroid isthmus must be in addition to the difficulty encountered in applying the instrument cause excessive tension on the isthmus of the gland or impinge the jaws of the instrument into the adjacent tissues of the neck.
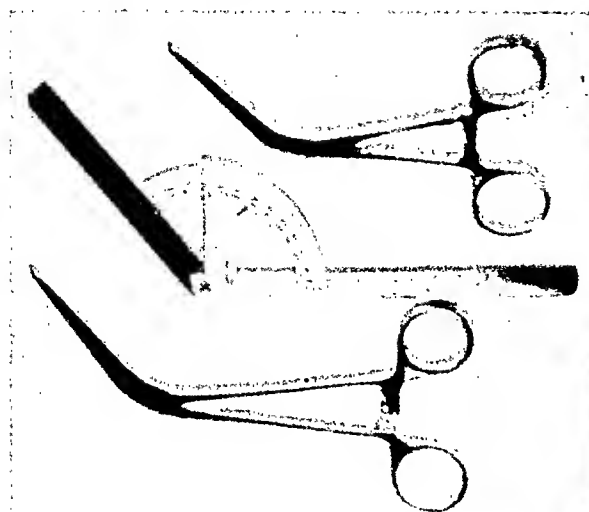
Fig. 1. Forceps with modified protractor; angle of 50 degrees chosen to allow for finger grips on handles of instrument in addition to normal angle formed by the neck on the chest.
Fig. 2. Patient in position for operation; note angle of clamp coinciding with anatomical angle formed by the trachea on the manubrium sterni.

Instrument Design. With this information at hand, an instrument was devised with an angle of 50 degrees made by the jaws on the handles and in line with the handles. The center of angulation is located at the center pin of the joint, a smooth curve is formed and limited to the joint portion of the instrument. (Fig. 1.) This permits approximation of the jaws and handles of the instrument in a normal manner and yet retains the necessary stability of the joint. A second point of merit in design is the longitudinal arrangement of the serrations of the jaws with superimposed transverse serrations at the distal ¼ inch. The longitudinal lines prevent the grasped tissue from slipping from the side of the instrument while the terminal transverse lines prevent tissue slipping from the end yet avoiding the disadvantages of a tooth at the end of the jaws. The opposing margins of the jaws are rounded to avoid the sharp angular effect on compressed tissue; this reduces the likelihood of tearing or cutting the tissues held by the instrument. The value of an angulated instrument such as this in thyroid surgery is obvious. Principally, it allows for ease in application to the thyroid isthmus without undue or dangerous traction on the trachea. (Fig. 2.) Once the isthmus is divided and the trachea is free the operation may proceed at the surgeon's leisure and with a greater degree of safety to the patient.

TECHNIC FOR SUBTOTAL THYROIDECTOMY

Anesthesia and Positioning. The patient is placed upon the operating table in a supine position with the foot of the table 10 degrees lower than the head to allow maximum drainage of the venous system of the head and neck. The patient is given a general anesthetic of gas, oxygen and ether. A small pillow is placed beneath the shoulders to allow maximum extension of the head, thereby accentuating the thyroid region and increasing the distance from the suprasternal notch to the inferior surface of the mandible. The skin is prepared with ether and tincture of mercuric chloride over a wide area. A fluffed towel is placed on either side of the patient's neck, and the patient is draped exposing the neck region from the suprasternal notch to slightly above the hyoid bone.

Skin Incision and Flap. A collar type skin incision is made at the desired level, usually about 3 cm. above the suprasternal notch, following the wrinkles of the neck

which places the incision in the normal cleavage line. This may be marked with a string. The skin incision is deepened through the platysma muscle and superficial layer of the deep cervical fascia, extended laterally by blunt dissection and between the strap muscles of the neck is divided in the midline and the muscles are freed separately from the surgical capsule of the thyroid gland by curved Mayo scissors and blunt dissection; the sternothyroid is identified, brushed aside as part
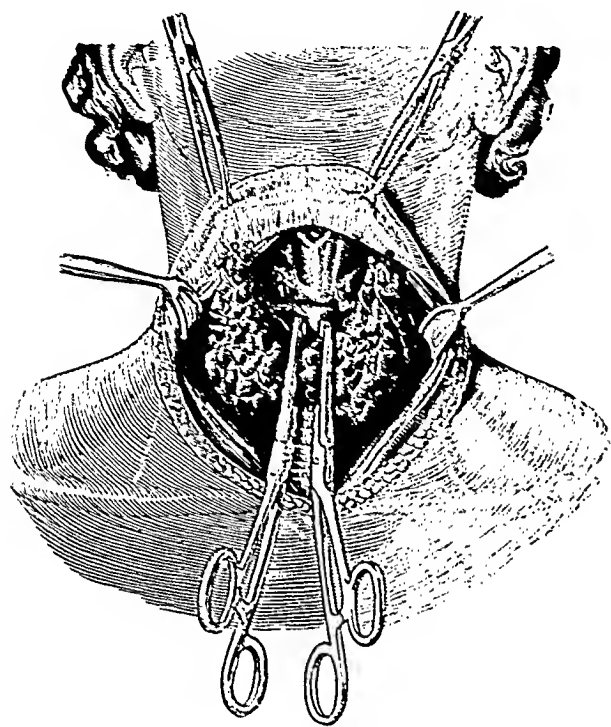
Fig. 3. Drawing from photograph taken at operation; isthmus clamps in position preparatory to division of the thyroid isthmus *en masse.*
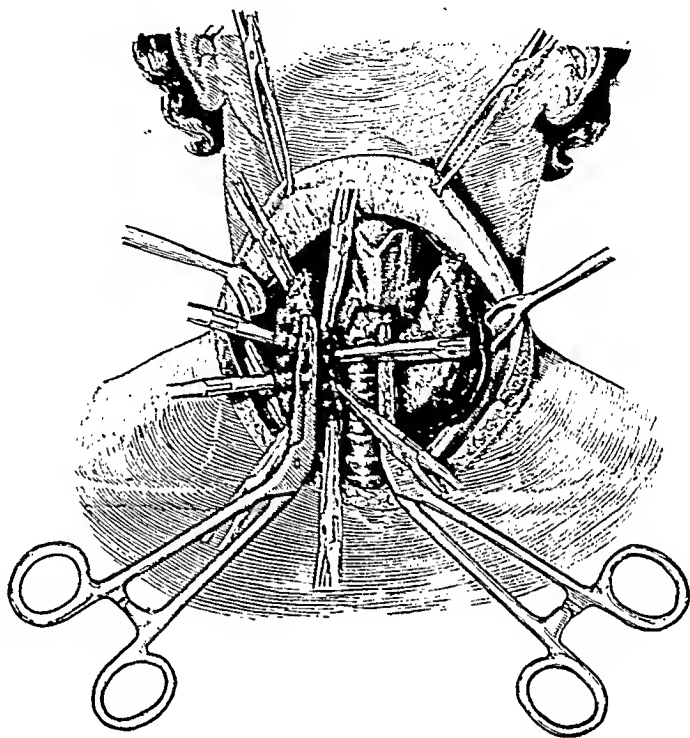
Fig. 4. Drawing from photograph taken at operation showing exposed anterior portion of trachea on rolling the isthmus clamps to the sides. Kocher clamps are placed to outline portion of thyroid to be removed by wedge-shaped excision after ligature of superior thyroid vessels. Clamps are removed as continuing suture of the capsule progresses.

cutting all layers simultaneously with a curved Mayo scissors. The anterior jugular veins are clamped and divided. The wound margins are draped with moist wash clothes and secured at either side of the neck with Smith hooks or Michel clips. All vessels clamped and divided are ligated with black silk No. C. The upper flap is freed by blunt dissection in the avascular tissue plane and reflected to the level of the suprathyroid notch. A blunt rake retractor is placed to retract the upper flap. The anterior borders of the sternocleidomastoid muscles are freed, permitting greater lateral retraction if necessary.

Exposure and Division of the Thyroid Isthmus. The middle layer of the deep cervical fascia forming the fascial bridge of the capsule and allowed to retract. This allows the gland to rise in the operative field giving greater exposure to the surgical capsule of the gland. The pretracheal fascia is divided above and below the thyroid isthmus and specially designed clamps (Kuker) are carefully introduced between the isthmus and trachea, the isthmus is clamped on either side of the midline with these instruments and divided *en masse* with a scapel between the clamps which further mobilizes the gland. (Fig. 3.) The handles of the angulated clamps are rolled laterally, peeling the isthmus from the anterior half of the trachea thus relieving any pressure produced by the isthmus and any tracheal compression which may have existed. (Fig. 4.) Resection of the lateral

lobes can progress with a greater degree of safety and without danger of respiratory embarrassment due to manipulation of the gland.

Management of the Superior Pole and Resection of the Gland. The lobe being removed is grasped with a Lahey clamp, pulled down with slight traction, lifted into view and rotated medially. The superior pole is freed by blunt dissection, and a curved Kelly or aneurysm needle is passed beneath the pole into which a black silk No. 1 ligature is caught and drawn about the pole and tied. A Kelly clamp is applied to the pole on the gland side of the ligature and the pole divided between clamp and ligature. With the lobe free and well exposed, the operator decides the amount of gland to be removed and outlines the remaining portion by application of Kocher clamps to vessels about the periphery of the gland. (Fig. 4.) The gland is cut away by shaving the clamps in a wedge-shaped manner and by so doing a dangerous area is avoided. Any vessels not caught from the periphery are caught as the gland is removed. This procedure leaves a portion of the gland along the posterior portion of the capsule. The recurrent laryngeal nerve is not visualized.

Suturing the Capsule. Instead of ligating each vessel and bleeding point individually, a continuous criss-cross suture of chromic No. 00 catgut is used and the clamps removed as the suture progresses. On the return suture an occasional bite of the tracheal fascia is included.

A dry sponge is placed in the side just completed and the same procedure is carried out on the opposite side. The operation is completed, sponges are removed, both sides are thoroughly inspected for hemostasis, and if the field is found to be dry, the wound is closed in anatomic layers without a drain.

Closure. The sternohyoid muscles are approximated in the midline with three to four interrupted sutures of black silk No. 1. Several subcutaneous interrupted sutures of black silk No. C are placed in the flaps which serve to approximate accurately the skin margins. The skin is closed with Michel skin clips placed about 3/8 inch apart. A dry dressing is applied and secured in place by one or two straps of adhesive which suspend from the back of the neck, cross and attach on the chest anteriorly.

COMMENTS

While cognizance is taken of controversy and differences existing at the present time on many of the technical aspects of thyroidectomy, we shall confine our comments to the surgical principles of the procedure as previously outlined.

Positioning of the patient is important both from the standpoint of immediate exposure and of postoperative comfort. Venous drainage of the neck is afforded with the head elevated. An interscapular rest or sandbag permits sufficient extension to give maximum exposure. Overextension of the neck gives rise to considerable postoperative discomfort in the occipital-cervical region.

General anesthesia is routinely used. A safe anesthetic is oxygen, ethylene and ether, and these are ordinarily preferred to other agents. An endotracheal tube may be employed; however, a tube gives rise to postoperative tracheitis, sometimes very severe, with resulting pulmonary complications. We believe that the dangers of not using a tube are, in the main, obviated by dividing the isthmus early, a step greatly facilitated by angulated isthmus clamps.

Outlining the proposed incision with a piece of suture material against the neck is an aid in making an incision which conforms to the natural skin lines in the neck. Bleeding is reduced to a minimum and the procedure expedited if after an initial skin incision with a knife, scissors are put through the platysma in the midline down into the avascular plane, inserted laterally and spread, and the skin and platysma tented with the scissors away from the underlying sutures and cut. Skin flaps are developed by blunt dissection and cleavage

planes are carefully followed. Better exposure is secured by partial freeing of the sternocleidomastoid muscles and division of the sternothyroid. There is no harm in leaving the divided ends unsutured. Because of the higher insertion of the sternohyoid, this muscle can be retracted satisfactorily except in very difficult cases. The sternothyroid on the other hand is in close contact with the superior polar vessels.

Division of the pretracheal fascia above and below the thyroid isthmus permits introduction of the specially designed clamps between the isthmus and trachea. The isthmus is divided between the clamps and the lobes freed, thereby relieving any possible tracheal compression or respiratory embarrassment and also permits access to the trachea in readiness for any eventuality.

The clamp described is specifically designed for the isthmus. Just as thoracic, abdominal and neurologic surgery require armamentaria peculiar to the various specialties, so does the anatomic configuration of the neck necessitate special instruments to accomplish efficiently certain phases of thyroid surgery. The thyroid gland is held down by the sternothyroid muscles and isthmus along with its attachments. The lobes of the gland, by the division of the isthmus after the sternothyroid muscles are removed as part of the capsule,

become freely mobile, and this mobility allows the gland to be put under tension in any direction thus simplifying all the further steps in excision or resection of the gland.

The gland is removed with the exception of a v-shaped portion along the posterior capsule. This portion of the posterior capsule protects the inferior recurrent laryngeal nerve and the parathyroid glands and is not molested. The nerve is not demonstrated routinely.

A continuous suture incorporates the lateral and medial aspects of the capsule, along with the tracheal fascia, and is a very efficient hemostatic suture.

Closure is effected without drainage. Excellent cosmetic results are obtained with subcutaneous, fine black, silk sutures and Michel clips for the skin, one-half of which are removed in twenty-four hours and the other one-half in forty-eight hours.

SUMMARY

1. A simplified technic for thyroidectomy has been presented.

2. An angulated thyroid isthmus clamp has been introduced.

MANAGEMENT OF RECURRENT VARICOSE VEINS*

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A GREAT deal has been written about the initial treatment of varicose veins but little has been written about the treatment of recurrent varicosities. I would estimate that approximately one of every five patients who consult me for varicose veins have had some form of previous surgical treatment for this condition. Each of these patients presents a highly individual problem and the treatment must be based entirely on the reason for the recurrence. Heyerdale[1] and the writer have previously pointed out a number of the factors which were existent in recurrence of varicosities following treatment. We stressed the fact that when incompetency of the saphenous system of veins existed, and when simple injection treatment was carried out that a recurrence could be expected in most every patient. For this reason we were primarily interested in the factors which existed in recurrence when previous surgical treatment had been undertaken. At this time it was our belief that the majority of varicosities could be adequately treated by a combined division, ligation, and injection of the great saphenous vein at the saphenofemoral junction together with separate division and ligation of the uppermost tributaries. When incompetency of the lesser saphenous vein existed, this was also ligated just posterior to the knee. Subsequent injections were carried out to completely obliterate the remaining patent varicosities. There is no question in the author's mind that if this procedure is adequately carried out a great percentage of the patients with varicose veins can be properly treated.

Many of the recurrences seen today could be prevented if the many variations in the anatomy of the great saphenous vein and its tributaries at the fossa ovalis are recognized and adequately treated. Heyerdale[2] and the author have previously described a technic which has made it possible for us to expose adequately the incompetent great saphenous vein and its various tributaries. In many instances, recurrences were secondary to failure to ligate the uppermost tributaries with a resulting shunt around the site of ligation of the saphenous vein. In other instances the lateral or medial superficial femoral vein had been mistaken for the saphenous vein with resulting persistence of incompetency of the great saphenous vein following its supposed ligation. In others the saphenous vein was ligated at too low a level. In some instances anatomic variances in the tributaries was the explanation for the recurrence. In a few it was our belief that had a segment of the ligated saphenous vein been excised recurrence might have been prevented.

There are a number of other factors which could be mentioned, but of most importance has been the lack of adapting the original operation to the type of varicosities present and failure to consider the individual patient and his environmental problem. It was the author's belief at the start that most patient's varicosities could be properly treated by performing the aforementioned great and lesser saphenous vein ligation followed by an adequate program of sclerosing therapy. After observing these people throughout a number of years it became obvious to him that in a certain percentage it was almost impossible to completely eradicate these varicosities by this method. In another group permanent symptomatic and cosmetic results failed to be obtained. In still others this satisfactory

result was obtained until the environmental or physical status of the patient changed. For example, the patient became pregnant. In still another group varicosities developed in portions of the venous system which had not been involved or previously treated.

From the patients' standpoint all individuals who developed varicose veins following any previous surgical treatment for such a condition must be classified as having recurrent varicosities. It is true that according to the author's grouping, a number of these individuals surgically should be classified as having (1) persistent varicosities, (2) recurrent varicosities and (3) newly developed varicosities. This is not practical so it is obvious that we are dealing with individuals who have had inadequate surgery at the time of the first treatment; individuals who have had adequate surgery but who as a result of either time, physical or environmental problems have had recurrences, or individuals who have developed new varicose veins. As one observes more and more of these people and studies the familial tendency he can predict the possible future course of his patient with varicose veins before any treatment is undertaken. With this in mind, the author selects with great care the operation, namely, a high ligation combined with sclerosing therapy; a high ligation together with segmental ligations; a complete or partial stripping operation; a classical high and low ligation together with sclerosing therapy or a ligation with segmental excision. In the past I believe that too little attention has been paid to the lesser saphenous vein, and a number of individuals that I have treated for recurrences had these because of incompetency of this vein.

It has been my observation that by selecting the operation which will most completely eradicate the patient's present, as well as his potential problem, that a number of the aforementioned recurrences could be prevented. In every instance the operation should be combined with sclerosing therapy. It has been my policy to look at all cases of recurrent varicose veins as untreated varicosities, and to treat these patients as was indicated anatomically. In some instances a little sclerosing therapy will be sufficient. In others if the original high ligation has been inadequate, it will be necessary to repeat this. If one or two communicating veins have become incompetent, a ligation of these, perhaps together with segmental stripping, may be indicated. In others a ligation of the lesser saphenous vein will suffice. In many of the recurrent problems, if it has been otherwise reasonable, I have been employing the complete stripping operation with removal of the entire great saphenous vein and, if indicated, the lesser saphenous vein.

In attempting to select the desired operation for these individuals there are a number of factors which influence me. If the patient is young and has already had considerable trouble from extensive varicosities with unsatisfactory results from a high ligation, there is nothing short of a stripping operation indicated. If there is a family history of considerable difficulty from varicosities and the patient has a fairly advanced condition, a stripping operation should be considered. Of course, in order to perform a stripping operation a general anesthetic, preferably spinal, is necessary. This means that the patient must be considered a minimal anesthetic risk. One must be careful to evaluate all of the associated vascular problems present as well as all of the general problems. Primarily, the treatment of varicose veins should be based on a symptomatic result rather than cosmetic. There are certain patients who must expect to retain a few varicosities if these are not producing symptoms. I do not mean to imply that everything reasonable should not be done to obtain cosmetic improvement but, for example, if one has, as I have at the present time, a patient aged seventy who has stasis ulcer associated with recurrent varicose veins, the indicated surgical treatment is that which will produce complete healing

of the ulcer and relieve the patient of his symptoms but which may not entirely eliminate all of his varicose veins. In this case I performed a segmental ligation of the saphenous veins, and even though his ulcer has been open for more than two years it is now healed at the end of three and one-half weeks. The patient who has large varicosities, who has had an old phlebitis which is not active at the present time, who has had much sclerosing therapy, who has had an ulcer which is now healed, or who has had an adequate high ligation with subsequent sclerosis and now has recurrent varicosities, is a candidate for a stripping operation. If the ulcer is active or if the stripping operation is considered too great a procedure for these individuals, a segmental ligation of the saphenous veins with some possible local stripping must be considered. This procedure, in contrast to the stripping, can be done entirely under local anesthesia.

When the varicosities are minimal and an inadequate high ligation has been previously performed with little or no subsequent sclerosing therapy, I perform a high ligation with sclerosing as previously mentioned. In others, with varicosities slightly more advanced and with a definite palpable saphenous vein below the knee, I usually perform a high and low ligation with subsequent sclerosis.

SUMMARY

For practical purposes all varicose veins which develop subsequent to any surgical procedure have been classified as being recurrent. These are considered and treated as primarily untreated varicosities. The operation to be performed is carefully selected for each patient and may be a high ligation with sclerosing therapy; a high ligation with segmental ligations; a complete or partial stripping; a high and low ligation with sclerosing therapy or a ligation with segmental excision.

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PROCIDENTIA

A NEW OPERATION TO CURE THE "REMAINING" PROLAPSED CERVIX OR VAGINAL HERNIA

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FOR the past thirty-five years I have made a careful study of the problem of prolapse of the uterus, bladder and culdesac tissues. I have written on this subject before[1-4] and in these writings have described certain new and original procedures that have produced most excellent results. In 1944 I last reported the results of a sufficient number of cases to justify conclusions and make valuable statistics. That study showed that the satisfactory results (to patient and doctor) obtained were about 99 per cent. Many have been added since, all private patients, and the results are still the same.

GENERAL CONSIDERATION

Subtotal Hysterectomy (Abdominal). The reason for mentioning this operation is to show its relation to the prolapse problem. I believe that many prolapsed cervices would not occur if a technically good abdominal subtotal hysterectomy was done at first. I mean that if the cervix is adequately and properly suspended (provided prolapse is not already present), many of the later prolapsed cervices would not occur. That can be done in only one manner and that is to bury the round ligaments into the open cauterized (Percy cautery) cut end of the cervix. This must be done before the cervix is closed. More than 90 per cent of surgeons and gynecologists, I have observed, still close the cervix and then attach the ligaments to the top. *This produces nothing but an adhesion.* Adequate suspension may be done in any manner the operator chooses, as long as the cervix is closed over the ligaments placed under fair tension. In my clinic I have evolved a

simple method of doing this with a single continuous suture called the "crossover mattress technic." (Fig. 1A and B.)

I cannot conceive of a more adequate suspension than this and it has been reduced to such a degree of simplicity that I

A   B
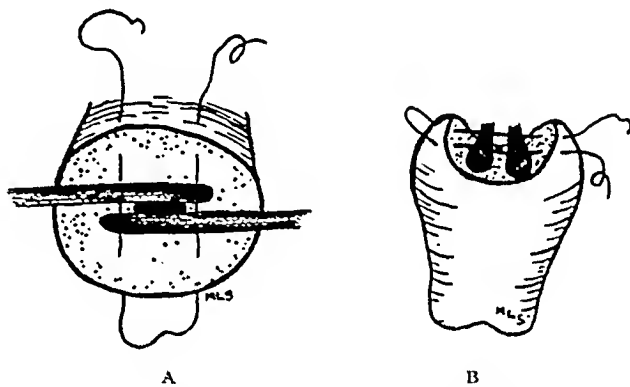
FIG. 1. A, shows the single suture (double No. 2 chromic) technic for making an adequate suspension of cervix. Tubes are not included, only round ligaments. This is called the "cross-over mattress technic." B, transverse view shows graphically the position of round ligaments to be buried in cervix before tying. Bladder flap covers this area with a single suture if fastened low on posterior cervix.

have done scores of those operations, including a general exploratory of abdomen with appendectomy in less than fifteen minutes. I mention this only to emphasize the simplicity of the abdominal subtotal hysterectomy with adequate suspension of the cervical stump.

Dealing with the Actual Prolapse. We divide the prolapse into (1) first and second degree which permits the cervix to come within 1 inch of the vaginal outlet when "squatting." (2) The third degree is at the outlet. (3) The fourth or nth is any degree beyond that. I want my readers to understand clearly before I proceed, that total vaginal hysterectomy plays no part in this surgical correction. I do not believe that

vaginal hysterectomy has ever been the proper procedure for prolapse.

Vaginal hysterectomy is a destructive procedure rather than a constructive one. It removes the only solid substantial tissue to anchor or support, viz., the cervix.
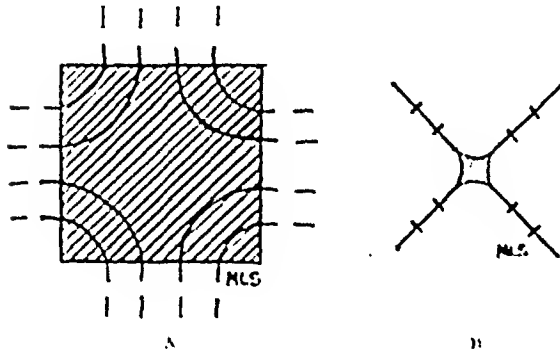

Fig. 2. A and B, method of excising and suturing the redundant cul-de-sac or vaginal hernia tissues by abdominal route (cervix may or may not be present). The opening in the center is to receive the round ligaments as in Figures 3, 4, and 5, except all are done by abdominal route unless cervix is present. If cervix is present, do interposition of cervix by vaginal route after abdomen is closed.

After the cervix is removed there is nothing left but a hole. Converting this hole into the semblance of a vaginal canal suitable for coitus and giving a support to the anterior wall and bladder (cystocele) is a problem that has never been satisfactorily solved. As many as 1,500 of these operations have been done by a single operator but the results are far below 99 per cent. Several gynecologists report an average of 10 to 15 per cent of poor results. Again, nearly every operator has his favorite technic of doing his vaginal hysterectomy, which in itself justifies the conclusion that none of the technics is satisfactory. The cervix should be removed only rarely either by the vaginal or abdominal route. Cystic cervices may all be cleared up with the Percy cautery or fulguration at the time of operation. Surgery is not the treatment of choice of cancer of the cervix. Incidence of cancer of the remaining cervix should be less than on unoperated women if the cervix receives the proper cautery treatment at the time of surgery.

Removal of the cervix predisposes to cystocele. This seems to leave few, if any,

specific indications for removing the cervix. Total hysterectomies take longer, are more dangerous to the patient, cause more danger of infection and give poorer support. Also operative and postoperative complications are more frequent. We, as teachers and instructors, should recommend to our students and postgraduate audiences the best procedure that will be safe in their hands. This point is frequently overlooked. Most of our series of vesicovaginal fistulas have followed total hysterectomy but few, if any, subtotal hysterectomy.[5,6]

SURGICAL TREATMENT OF PROLAPSE

I believe, and I have ample evidence to substantiate my opinion, that the interposition operation is the best technic to cure. It is one hundred per cent better because the uterus closes the opening through which the bladder protruded leaving room only for the urethra. It also removes all traction on the bladder neck and remedies leaking. By its extreme antiversion it remedies the second degree prolapse and leaves the cervix pointing toward the sacrum.

The Nth Degree Prolapse. In this surgical entity I will describe briefly the[2,3] Chaffin prolapse operation or specifically the *vaginal subtotal hysterectomy with transplanting round ligaments.*[1-4] I have improved slightly the "anchor" since my publication in 1944 by puncturing the posterior vaginal wall and passing a forceps up through the hole behind the cervix, grasping the ends of the ligaments, broad and round, and pulling the ends well down into the vagina, holding them tightly while suturing. Ends ½ to 1 inch long need not be excised as they will shrink into a hard, fibrous knot of tissue to assure a continuous support of the posterior vaginal vault. The hole is made close to the posterior surface of the cervix. (Figs. 3, 4, and 5.)

If there is a large redundant posterior vaginal wall, so-called vaginal hernia, we resect a portion of the redundancy by excising a rectangular piece of tissue ade-

quate to reduce the redundancy and close it by the four-corner suture method. (Fig. 2A and B.) We then pull the ligaments through and anchor them. The remainder of the operation is to do a typical classical

1. Prepare patient's abdomen and vagina.
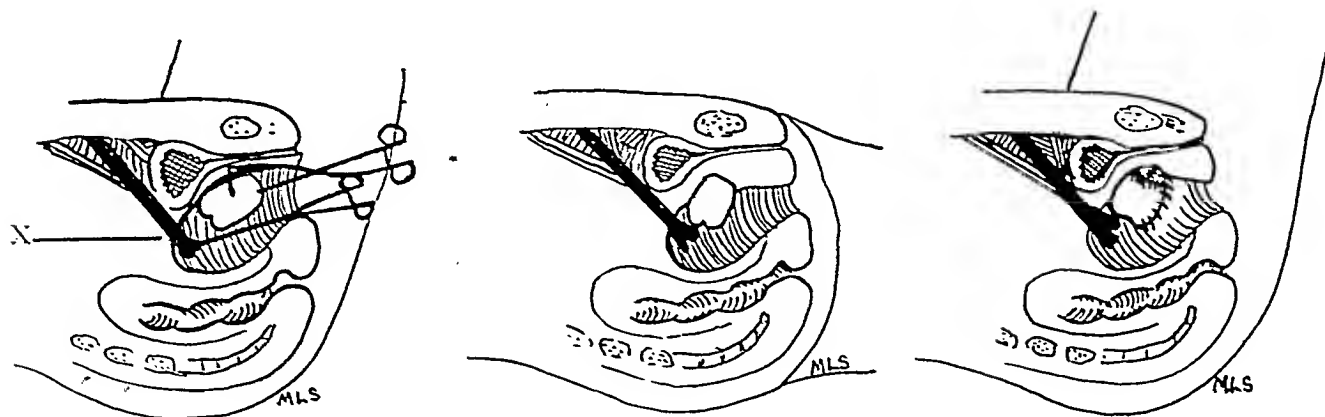
2. Trendelenburg position— 40 to 45 degrees.

FIG. 3. Sagittal section shows graphically the round ligaments being pulled into the vagina through a hole behind the cervix. Sutures are placed at X, free ends protruding. Corpus has been removed vaginally thereby making round ligaments available for transplanting. This is the Chaffin operation for procidentia.
FIG. 4. Shows the round ligaments sutured into the fenestrated vaginal opening behind the cervix. The next step is the interposition suture through the pubovaginal fascia and closed end of cervix to close the "cystocele" space.
FIG. 5. Shows same as Figure 3 after ligaments have been sutured and the swelling and edema that follow, thereby assuring a permanent attachment. Cervix (internal os end) is closed and sutured beneath urethra as in interposition operation.

interposition of the closed cervical stump. A perineorrhaphy completes the operation.

REMAINING PROLAPSED CERVICES FOLLOWING A PREVIOUS ABDOMINAL SUBTOTAL HYSTERECTOMY

During my nearly thirty years as Professor of Gynecology, I made it a point to ask all visiting gynecologists what to do with the prolapsed cervix. I do not recall ever getting a direct answer. That prompted and stimulated me to go to work on the problem. I believe that I have now solved it completely and have about sixteen cases all 100 per cent supported and without a failure. I believe that justifies a report and description of the technic.

I might dispose of the problem by saying that we do the Chaffin operation, one-half by the abdominal route and one half by the vaginal route. That is what we really do but in the following paragraphs I will attempt to make it a little clearer than I did in my last publication.

3. Suprapubic laparotomy incision; wound towels—five-yard pack to empty pelvis.

4. Identify and grasp cervix with tenaculum.

5. Dissect bladder away from cervix and well down the anterior wall to vaginal wall.

6. Identify round ligaments and mobilize.

7. With tenaculum grasp culdesac posterior and close to cervix.

8. Incise (fenestrate) at this point into vagina.

9. Insert round ligaments into the opening while pulling up on vagina to make ligaments taut.

10. Suture well at these insertions with No. 2 chromic catgut.

11. Leave cervix free in the anterior position and cover with bladder flap.

12. Suture bladder flap at about the point of ligament insertion or farther if it will reach.

13. Close abdomen.

14. Lithotomy position; Gilpi retractor.

15. Incise anterior vaginal wall and cervix will be seen at once in this field.

16. Place classical interposition suture through pubovaginal fascia and thereby anchor the cervix under the urethra.

17. Close incision and operation is completed.

18. Perineorrhaphy if necessary.

19. Vaginal pack for one to two days.

The operation for the prolapsed vaginal vault in absence of the cervix is done entirely by the abdominal route: (1) Open the abdomen in 45 degree Trendelenburg position and identify the round ligaments which are usually retracted. (2) Grasp the apex of vaginal vault (nurse will push a blunt instrument in vagina to identify the "high point") with a tenaculum. Resect, if necessary, (Fig. 2A and B) and insert round ligaments (Figs. 3, 4, 5.) and suture. The bladder will also be supported by this technic. (3) Insert vaginal pack for a few days.

SUMMARY

1. A simple abdominal hysterectomy is described with an adequate suspension of the cervix.

2. Cystocele cure is assured by the interposition operation.

3. An nth degree uterine descensus is completely remedied.

4. The problem of the remaining prolapsed cervix is treated by a new original method with 100 per cent cures.

5 Vaginal hernia after total hysterectomy is cured by the technic herein described.

CONCLUSION

1. Abdominal subtotal hysterectomy is nearly always indicated for the usual pelvic disorder and more attention should be given the support of the cervix. Sew ligaments *into* not *onto* the cervix.

2. Vaginal subtotal hysterectomy gives far better physiologic and anatomic results than total vaginal hysterectomy. The statistics justify this statement. Vaginal hysterectomy does *not* cure the prolapsed uterus; it only removes it.

3. The vaginal subtotal hysterectomy is easier to do than the vaginal total. The average time it takes is about forty minutes including perineorrhaphy, the minimum fifteen minutes.

4 Approximately sixteen patients with prolapsed cervix and vaginal hernias have been operated upon by this technic and all have been cured.

5. To improve the over-all results in dealing with the procidentia problem, these two technics should be adopted by all surgeons doing this work, thereby reducing the 10 to 15 per cent failures reported by others.

6. The criteria for cure are: (1) A vaginal canal of normal depth permitting satisfactory coitus; (2) substantial anterior wall with no bladder bulging in the squatting position; (3) the culdesac occupies a high point to examining finger in the supine and squatting positions and (4) a vaginal canal normally lubricated by a normal cervical secretion is assured.

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USE OF CURARE IN THE ANESTHETIC MANAGEMENT OF THE PROFOUNDLY SEDATED PATIENT*

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THE purpose of this paper is to describe the use of curare in patients premedicated with large doses of morphine and scopolamine. The study is based on an evaluation of 446 cases; one hundred of these have been subjected to analysis.

The sedation in this series comprises two or three injections of morphine and scopolamine. Basal narcosis and total amnesia were the primary aims of this heavy medication. Although the amount of drugs used was regulated by careful observation of respiratory and pulse rates, it proved difficult to anticipate the individual responses of the patients, and many unusual problems were encountered by the anesthesiologist.

Upon arrival at the operating room the patients were drowsy or unconscious. A large percentage of them manifested an ideal reaction to the drugs and were easily managed by the anesthesiologist. They had normal or only slightly depressed respirations and normal pulse rates. Invariably, however, all of these patients were in a state of generalized increased muscular tone, and most of them showed a ballooning and loss of tone of the intestines. These two factors gave the anesthesiologist particular concern in the intra-abdominal cases.

A number of these patients were flushed, with a florid, slightly cyanotic appearance, a rapid bounding pulse of 110 to 160 per minute, showing evidence of cerebral irritation due to the large dose of scopolamine. They were readily aroused to slight or moderate delirium upon even the mildest stimulation.

Another group evidenced a drug imbalance with a preponderance of morphine depression signs. The respirations were decreased in rate, occasionally falling as low as 1 to 2 per minute. It often seemed that the surgeon could proceed with his operation without the aid of an anesthetic agent. However, patients forcibly resisted painful stimulation and lacked the cooperative ability accompanying consciousness.

Establishing surgical anesthesia and adequate relaxation in these semi- or completely unconscious, rigid patients proved a hard task with the anesthetic gases available. Cyclopropane was found to be unsatisfactory because it enhanced the already existing respiratory depression. The "controlled respiration" technic was precluded because of the possible difficulty in restoring the automaticity of respiration.

Ethylene-oxygen or nitrous-oxide-oxygen did not provide sufficient muscular relaxation, and the slow rate of respirations made it difficult to add enough ether to obtain the desired anesthetic depth.

Since November 1, 1944, curare has been employed in the anesthetic management of these heavily premedicated patients. This method of combating the undesirable muscular tension and of obtaining adequate relaxation has met with singular success and has become routine whenever large doses of morphine and scopolamine are used for preanesthetic narcosis.

METHOD OF ADMINISTERING MORPHINE AND SCOPOLAMINE IN DIVIDED DOSES AS PREANESTHETIC MEDICATION

The patients were premedicated in the manner described by Metz in 1936.[1] Two and one-fourth hours before the scheduled

time for surgery the first hypodermic injection of morphine and scolopamine was given. The dosage was morphine 16 mg. (gr. ¼) and scopolamine 0.65 mg. (gr. ¹⁄₁₀₀) for normal adults of good vigor. The first dose was reduced if the patient was undernourished, debilitated or over sixty years of age. The second hypodermic was given forty-five minutes later. This consisted of morphine 16 mg. and scopolamine 0.65 mg., or morphine 10 mg. and scopolamine 0.5 mg., depending on the patient's vigor. The third dose was usually morphine 10 mg. and scopolamine 0.5 mg., administered forty-five minutes after the second dose, but was omitted if the patient was unconscious and did not respond to stimulation. One-half of the last dose was given if the patient was aroused with difficulty, the pulse rate below 100 and the respiratory rate above 10. In elderly patients the nurse was instructed to notify the anesthetist regarding the pulse and respiratory rate and the state of drowsiness before the second dose. In all cases the anesthetist was notified before the third hypodermic injection.

METHOD OF ADMINISTERING CURARE

The preparation of curare used was intocostrin,* and the concentration was 20 units per cc. The curare was administered by means of a simple infusion, consisting of a 5 cc. syringe, a two-way stop cock and a foot length of non-compressible small caliber tubing attached to a No. 19 gauge needle.

Routinely a 20 unit dose of curare was given intravenously immediately before the anesthetic mask was placed on a patient's face. This caused sufficient muscular relaxation to allow a quiet induction with ethylene or nitrous oxide and permit adequate oxygenation. When surgery was extra-abdominal, the original 20 units were complemented by 20 units for the average case. For the operations which required deep muscular relaxation, the primary dose was followed by 40 units

* Extract of curare, Squibb.

five minutes before the anticipated time of the peritoneal incision. When relaxation was insufficient, an additional 20 units were infused and occasionally another dose was required for closure of the peritoneum.

TABLE I
LIST OF OPERATIONS IN THE SERIES OF ONE HUNDRED CASES

Type of Surgery	Percentage of Cases	
Intra-abdominal		
Cholecystectomies	24	
Hysterectomies	19	
Appendectomies	15	
Bowel resections	6	
Abdominal explorations	4	
Salpingectomies	2	70%
Extra-abdominal		
Rectal operations	13	
Herniotomies	10	
Orthopedic cases	5	
Bronchoscopy	1	
Removal of cervical polyps	1	30%

In all cases studied the following technic of administration of the inhalation agent was found optimal. The induction was accomplished with 3 liters per minute flow of ethylene or nitrous oxide and 1 liter per minute of oxygen. After approximately three minutes the anesthetic agent was reduced to 1 liter per minute and this 50-50 concentration, assuring adequate oxygenation, was continued throughout the surgical procedure.

RESULTS IN ONE HUNDRED CASES

Table I shows the division of 100 cases into types of surgery performed. Seventy per cent of the cases were intra-abdominal interventions, the highest number being upper abdominal surgery. The average duration of anesthesia per case was eighty-four minutes, the shortest anesthesia lasting nineteen minutes and the longest two hundred forty minutes.

The patients ranged in age from seventeen to seventy-nine years, the majority of the group falling between the fourth and fifth decades. Thirty-five per cent were males, sixty-five per cent females.

Table II divides the patients according to the anesthesia risk, using the standard risk classification advocated by the com-

mittee of Records and Statistics of the American Society of Anesthesiologists, Inc. Sixty-eight per cent were in excellent condition for surgery. The poorer risks included asthma, bronchiectasis, cirrhosis of the liver, severe cardiac disturbance and diabetes.

TABLE II

DISTRIBUTION ACCORDING TO RISK (ONE HUNDRED CASES)

Risk	Per Cent of Cases
I	68
II	25
III	3
IV	1
V	2
VI	1
VII	0

TABLE III

PREMEDICATION DOSAGE RECORD

Morphine Sulfate (Mg.)			Scopolamine Hydrobromide (Mg.)			Per Cent of Cases
3 doses..........| 16	16	16	0.65	0.65	0.65	1
	16	16	10	0.65	0.65	0.5	10
	16	10	10	0.65	0.5	0.5	19
	10	10	10	0.5	0.5	0.5	33
	10	10	8	0.5	0.5	0.43	4
	16	10	5.4	0.65	0.5	0.21	1
	10	10	5.4	0.5	0.5	0.21	1
2 doses..........| 16	16	....	0.65	0.65	....	4
	16	10	....	0.65	0.5	....	9
	10	10	....	0.5	0.5	....	5
	10	8	....	0.5	0.43	....	3
1 dose..........| 16	..	....	0.65	....	....	3
	16	..	....	0.43	....	....	1
	10	..	....	0.5	....	....	6

The amount of morphine and scopolamine used as the premedicant varied considerably. (Table III.) The maximum total dose was 48 mg. of morphine sulfate and 1.95 mg. of scopolamine hydrobromide. This amount was given to only one patient. Ten patients received 42 mg. of morphine and 1.80 mg. of scopolamine. Thirty-three per cent had a total dose of 30 mg. of morphine and 1.50 mg. of scopolamine. The minimum used was 10 mg. of morphine and 0.5 mg. of scopolamine which produced adequate amnesia and sedation in 6 per cent of the cases. The remaining fifty-one patients had amounts varying

between the above figures. Sixty-nine per cent of the patients received three hypodermic injections, twenty-one per cent two injections and ten per cent, one.

Table IV presents the amount of curare administered in this series of one hundred patients. Eighty-one per cent received 110

TABLE IV

DOSAGE VARIATION IN THE SERIES OF ONE HUNDRED CASES

Curare

Amount Used in Mg.	Per Cent of Cases
20–30	1
40–50	20
60–70	15
80–90	20
100–110	25
120–130	8
140–150	8
160–170	2
180–190	1 Total 100%

units or less of curare, with a maximal dose of 190 units in one case, and a minimal dose of 20 units in one case. The degree of muscular relaxation attained was attested by direct observations of the anesthetists and by information requested from the surgeons. Ninety-three per cent were graded as excellent, three per cent as fair and four per cent as poor. The latter group occurred early in the series and was due to insufficient curare. Rectal surgery required more curare than other extra-abdominal cases (often 80 to 90 units) due to the marked stimulation during dilatation of the anus.

There were few untoward respiratory effects from the small divided doses of curare. The respiratory rate was not reduced beyond the preanesthetic level. Often, in fact, stimulation from surgery seemed to increase the rate and maintain a better tidal exchange. There were four instances of intercostal paralysis lasting five to seven minutes, accompanied by rapid shallow respirations. These were treated by amplifying the tidal exchange by manual compression of the breathing bag. Two patients had diaphragmatic muscle paresis which lasted five minutes. One apnea of five minutes' duration responded to inter-

mittent inflation of the lungs. In no case was it necessary to use prostigmine as an antidote.

There were two deaths in this series of 446 cases. One patient (risk 4) expired on the fourth postoperative day with a diagnosis of carcinoma of the pancreas and cirrhosis of the liver; the other died on the fifth postoperative day with a diagnosis of coronary thrombosis. There was one postoperative pneumonia with recovery, and one coronary thrombosis which occurred on the seventh postoperative day. This patient also recovered.

Sedatives or analgesics were seldom necessary during the entire postoperative period and complaints of pain were minimal. Parenteral fluids were never administered in surgery and seldom during convalescence. The patients usually took fluids

TABLE V

SUMMARY OF EVERY TENTH CASE IN THE SERIES

Typical Cases

Case No.	Type of Surgery	No. of Hypodermic Injections	Amount of Curare Used (mg.)	Length of Surgery in Minutes	Time from Completion of Surgery to Alert Response to Questioning (min.)
10	Appendectomy	2	100	33	95
20	Appendectomy and exploratory lap.	1	100	92	10
30	Appendectomy	3	80	47	5–10
40	Hysterectomy and oophorectomy	2	100	110	85
50	Removal of semilunar cartilage	2	60	54	40
60	Appendectomy	3	40	60	10
70	Cholecystectomy	3	100	92	135
80	Herniotomy	3	40	62	115
90	Hysterectomy	3	60	97	200
100	Hemorrhoidectomy	2	40	47	20

COMMENTS

Psychologically speaking, this combined method had much in its favor. Most of the patients had no recollection of the trip to surgery; in fact, few remembered the second hypodermic injection. After the completion of the operation they responded alertly to questioning in from ten minutes to three hours. (Table v.) For the next few hours they dozed quietly, responding when disturbed, but sleeping the rest of the time. They were very enthusiastic about this type of basal narcosis, and their statements that "we never even knew we had been to surgery" pleased surgeons and relatives.

freely by mouth as soon as consciousness returned. The average length of stay in the hospital compared favorably with that following other types of anesthesia.

The pharyngeal airway was easily introduced in light planes of surgical anesthesia, without illiciting spasm of the jaw or vocal cords. When curare was not used, trismus of the masseter muscles was often initiated by attempts to insert an artificial airway.

Poor risk patients tolerated well the combination of heavy sedation and curare. The asthmatic patients showed no increase in bronchiolar spasm in spite of the large preoperative morphine dosage. Patients with liver, urinary and cardiac disorders were apparently unaffected by the anesthesia, and the diabetic showed no evidence of acidosis.

The total quantity of curare per case was less than that used when the anesthetic agents were administered without the heavy premedication of morphine and scopolamine.

SUMMARY

1. Curare has been used as an adjuvant to gas anesthesia following preanesthetic medication of large doses of morphine and scopolamine in 446 surgical procedures since November 1, 1944. One hundred of these cases have been subjected to detailed analysis.

2. Small divided doses of curare adequately secured the desired muscular relaxation in 93 per cent of the cases studied.

Seventy per cent of the cases were intra-abdominal interventions.

3. The total dose of curare per case was less than the amount required when heavy sedation was not used.

4. No contraindications to this method were noted.

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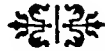
K. A. Kraft reports the use of intravenous procaine (1 to 1000 solution) in almost 900 operative patients in whom cyclopropane or other anesthetic was also given. He believes that this treatment prevents or/and effectively corrects any cardiac arrhythmias caused by cyclopropane and also makes it possible to obtain satisfactory anesthesia with a lesser amount of gas. Kraft has even used these intravenous procaine injections in the early postoperative period so that the necessity for opiates will be less. (*Richard A. Leonardo, M.D.*)

ANTERIOR DISLOCATION OF THE ELBOW WITH FRACTURE OF THE OLECRANON*

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ANTERIOR dislocation of the elbow is definitely a rare type of dislocation of the bones about this joint. Of the reported cases to date anterior dislocation alone is more common than anterior dislocation with fracture of the olecranon. A review of the literature up to the present time yields thirty-four reported cases, to which it is my desire to add another. The case to be reported is that of an anterior dislocation of the left elbow with fracture of the olecranon.

Although this type is not of the unusual anterior dislocation without fracture, only seven cases of dislocation with fracture have been reported up to this time. I agree with Cohn[1] and Jackson[2] that this condition presumably is not as rare as a review of the literature might indicate. Many cases have occurred no doubt but have not been reported. This case which is now added brings the total to thirty-five.. Twenty-seven cases with anterior dislocation without fracture and eight cases with fracture of the olecranon are included. (Table 1A and B.)

TABLE 1A

CHRONOLOGICAL ORDER OF REPORTED CASES OF ANTERIOR DISLOCATION OF THE ELBOW

1.	Evers	1787	15.	Staunton	1905
2.	Colson	1818	16.	Winslow	1913
3.	Leva D'Auvers	1842	17.	Cohn	1922
4.	James Prior	1844	18.	Tees	1923
5.	Monin de Mora	1846	19.	Tees & McKim	1929
6.	Guyat	1847	20.	Tees & McKim	1929
7.	Wittlinger	1848	21.	Simon	1930
8.	Ancelson	1859	22.	R. Bonn	1931
9.	Secreston	1860	23.	Christopher	1932
10.	Canton	1860	24.	Oddone	1932
11.	Gaussin	1861	25.	McKim	1939
12.	Hutchinson	1866	26.	Jackson	1940
13.	Fulton	1897	27.	Mathewson	1940
14.	Platt	1899			

This type of dislocation is barely mentioned by various authors in writing of injuries about the elbow. Callahan[3] and Chamberlain[4] mention the condition as very rarely occurring in dislocations about the elbow. Conwell[5] mentions anterior dislocation of the elbow, only to state that it is an extremely rare occurrence. He also

TABLE 1B

CHRONOLOGICAL ORDER OF REPORTED CASES OF ANTERIOR DISLOCATION WITH FRACTURE OF THE OLECRANON

1.	Richet	1839	5.	Mons	1877
2.	Morel Lavalee	1858	6.	Stimson	1899
3.	Richet	1859	7.	Batut	1910
4.	Rigaud	1870	8.	Strug	1946

states that the reduction accomplished is exactly the reverse of reduction of posterior dislocations.

In 1922 Cohn[1] published an exhaustive review of the literature and also reported an additional case. The literature in the nineteenth century is full of contradictions as to whether or not a forward dislocation at the elbow can occur without fracture. Boyer[6] stated in 1822, "We have never seen an anterior dislocation of the elbow complicated by a fracture of the olecranon." Gross[7] stated in 1859 that "anterior dislocation was believed impossible without fracture." Later events of recorded cases have certainly disproved both these assertions.

In 1931 Simon[8] also reported a review of the literature and added a case. Tees and McKim[9,10,11] have reported four cases on their service up to 1939. Jackson[2] in 1940 reported an additional case without fracture but complicated by a rupture of the brachial artery. Further evidence of the rarity of this condition is that in the x-ray service of Touro Infirmary, New Orleans, only two cases of this type have been seen during a period of twenty-five years, Cohn's[1] reported case without fracture in

* From The Department of Surgery, The Touro Infirmary, New Orleans, La.

1922 and the case herein reported with fracture of the olecranon. ·

MECHANISM AND PATHOLOGY

By far the majority of reported cases have occurred as a result of a direct blow upon the elbow. However, bizarre types of injuries may cause this type of disorder, as Gaussin's patient whose forearm was caught in a fly wheel. (This case was quoted by Cohn.[1]) However, most authorities do agree that a fall on the flexed elbow is the most likely cause of this type of dislocation. The pathological condition, as agreed upon by most authorities, is one of extensive lacerations of the ligaments about the joint, injury to the muscles about the joint and even going as far as separation of the muscles from the bony attachments. The resultant hemorrhage into the soft parts is severe and frequently calcification of the hematoma does occur even though accurate reduction has been accomplished.

The mechanism in those cases which have a fracture of the olecranon is readily understood. The resistance of the olecranon is removed so that the two bones of the forearm can be easily displaced forward. Actually these types of cases belong in a separate classification. Even so, the number of reported cases of this type are few.

The consequence of this type of fracture and dislocation can indeed be serious. The blood vessels about the elbow joint can be badly torn, possibly resulting in gangrene of the forearm and necessary amputation. Three such cases are reported by Couton,[12] Morel Lavalee[13] and Rigaud.[14] Injury to the brachial artery may go unrecognized. Marnheim[15] in 1934 and Eliason and Brown[16] in 1937 called attention to the fact that the vessels about the elbow joint can be lacerated in posterior dislocation of the elbow joints. In Eliason's series[16] one patient died and one case resulted in ischemic contracture. That injury to the vessels does not occur more often is indeed surprising and can only be attributed to the marked elasticity of the vessels. That this injury to the vessels does occur in anterior dislocations is evident by the report of amputations in the early reported cases. Recently, Jackson reported a case of anterior dislocation of the elbow with rupture of the brachial artery. Fortunately for the patient, this complication was recognized early, the area explored, the severed ends of the artery sutured and reinforced with a venous cuff. An excellent result was obtained. Mathewson,[17] in his discussion of Jackson's paper, mentions another case of anterior dislocation of the elbow, with rupture of the brachial artery, which was successfully sutured after reduction of the dislocation was accomplished.

If following reduction the radial pulse does not return, one should suspect a possible injury to one of the major vessels about the elbow joint or the presence of a massive hematoma. Operative intervention should immediately be instituted, the clot evacuated and, if a severed or injured vessel is found, either primary repair effected or simple ligation of the severed ends performed. It also must be kept in mind that the absence of a radial pulse following reduction may be due to arterial spasm which has occurred as a result of the trauma. If no marked swelling or hematoma exists to indicate a lacerated major vessel, an upper thoracic and stellate sympathetic block may be of value in terminating the arterial spasm.

If reduction cannot be readily accomplished, one should suspect that the triceps tendon has been lacerated and the distal end of the humerus had buttonholed through it. McKim[9] reports such a case in which operative reduction was necessary. Cohn's[1] case also needed operative intervention to accomplish reduction as there was tearing and buttonholing of the triceps. This case was further complicated by fracture of both bones of the forearm.

CASE REPORT

S. R., a white male, age eleven years, gave the following history. While riding a two-wheel
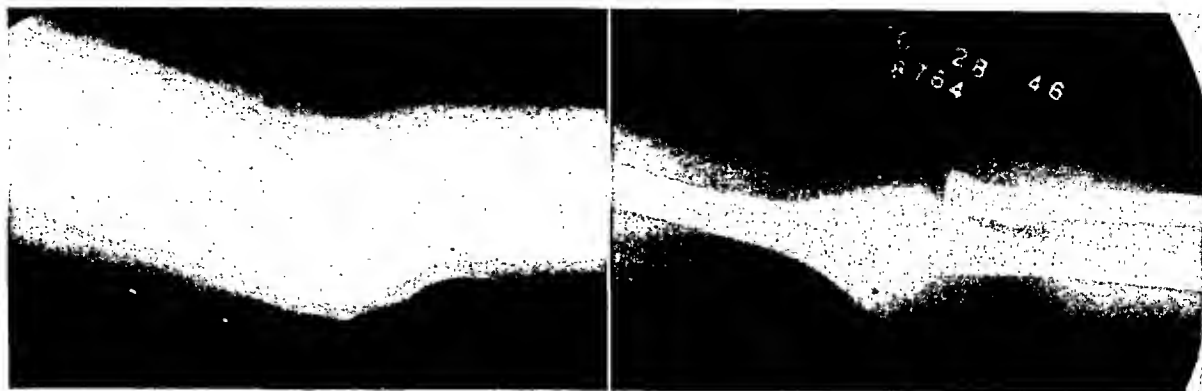
Fig. 1. Lateral view of left elbow, showing anterior overriding of the ulna and radius upon that of the distal end of the humerus. The epiphysis and the proximal fragment of the olecranon are seen remaining posteriorly.

Fig. 2. Anterior view of the dislocation and the anterior position of the distal ulna.

bicycle on a paved street, he struck a hole in the street with his front wheel and was thrown from his bicycle, his left elbow striking against the trunk of a tree. He immediately experienced severe pain in the elbow which was excruciating with the slightest movement. He was seen in the Emergency Clinic at Touro Infirmary. Physical examination revealed a well developed, well nourished white male child who was in acute pain. His left upper extremity was held in extension with the forearm in supination. He supported the left upper extremity with the right hand. There was a marked concaval deformity on the posterior surface of the left elbow. The anticubital fossa was full and bulged. The entire elbow was markedly swollen. The bony prominence of the olecranon was absent, however, and the external and internal condyles were in their normal relation. Any attempt, no matter how gentle, to flex the forearm caused severe pain. Any attempt by the patient to pronate or supinate the forearm elicited severe pain. The radial pulse was readily palpable although somewhat diminished in volume as compared with the opposite extremity. The fingers of the hand were readily moved. No sensory disturbance distal to the elbow was noted.

The entire left upper extremity was splinted and the patient was brought to x-ray. Roentgen examination of the left elbow revealed an anterolateral dislocation of the elbow joint associated with a fracture of the olecranon process of the ulna. An angular shell of bone

Fig. 3. Anterior view of the same elbow following reduction. Normal anatomical restitution of the fracture and dislocation is seen.

Fig. 4. Lateral view of the elbow with the forearm in 90 degrees flexion showing the normal relationship of the bones about the elbow restored.

comprising the superior margin and upper portion of the posterior margin of this process were detached, maintaining their relations with the humerus, while the major forearm fragments were carried forward. (Figs. 1 and 2.)

Under ethylene anesthesia reduction was accomplished in the following manner: With an assistant exerting countertraction, steady traction was applied to the forearm. The forearm was then suddenly flexed, with pressure being exerted in the anticubital fossa, and the ulna was reduced without difficulty. The reduction was confirmed by immediate fluoroscopy. (Fig. 3.) The forearm was placed in 90 per cent flexion, with the forearm in mid-pronation. The radial pulse remained palpable following reduction. An anterior and posterior plaster splint was applied extending to mid-arm.

Roentgen re-examination of the flexed left elbow in plaster revealed perfect reduction of the fracture dislocation of the elbow joint which was previously described. (Fig. 4.) The arm was allowed to remain in the plaster splint for a period of three weeks. The splint was then removed and daily physical therapy instituted. Complete extension and hyperflexion returned slowly. The patient was discharged on January 24, 1947, with full return of function.

CONCLUSIONS

Anterior dislocation of soft bones of the forearm at the elbow is rare. This reported case brings the total to thirty-five which have, to my knowledge, been recorded in the literature. This case, however, does not belong in the pure type of dislocation, as it has an associated fracture of the olecranon. The damage to the elbow joint can be great, and one must bear in mind the possibility of rupture of the brachial artery; early operative intervention can save the extremity as has been proved in the past. Spasm of the artery may have resulted from the trauma and has been reported.

Reduction, judging from the reported cases and my own, has not been difficult unless the humerus has been pushed through an extensive laceration in the triceps tendon. Mobilization should be started early, between two to three weeks

following reduction, unless there is an associated fracture of the radius and the ulna.

In the case reported an excellent functional and anatomic result was obtained.

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MIDDLE MENINGEAL HEMORRHAGE*

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EXTRADURAL hemorrhage of surgical proportions almost always results from injury to the middle meningeal artery. According to Munro it is present in 3 per cent of all cranial trauma. This important subject has been frequently and adequately discussed. However, in reviewing a recent series of our cases (Table 1) added data were found which we believe worthy of emphasis.

ETIOLOGIC CONSIDERATIONS

Our group of twelve patients were all males, ranging in age from seven to fifty-three years, with the surprisingly low age average of twenty-six.

The rapidity of development and the character of the clinical picture was related not only to the severity of the trauma but also to that part of the middle meningeal artery from which the hemorrhage arose. Thus six of our patients suffered relatively minor injuries such as a fall down the stairs, slipping upon an icy pavement, etc., and yet four of these had an extraordinarily explosive clinical course and a high mortality resulted in spite of what we considered prompt surgical attention. Our criteria of relatively minor cranial trauma are that the patients promptly and more or less completely recovered consciousness following the injury and there was little evidence of severe associated brain damage. Nevertheless when the main stem or a very large branch of the middle meningeal vessel was severed, the patient's life was threatened in a matter of hours.

Conversely six of our patients suffered what could be classified as severe cranial trauma: auto accidents, tree falling on head, etc., and in three of these the development of evidence of increasing tension was relatively slow. In each of these instances a relatively small branch of the middle meningeal vessel was involved. The severity of the associated cerebral trauma also served to mask, for a time at least, the evidences of increasing cerebral compression.

In all instances in which the point of trauma to the head could accurately be determined, the hemorrhage was found beneath this area. In most instances a horizontal linear fracture of the skull was recognized and proved to be the cause of the rupture of the artery.

SYMPTOMATOLOGY

The majority of our patients presented the classical picture of this disorder: an initial period of unconsciousness following the injury, succeeded by a lucid interval and a subsequent period of impairment of consciousness with focal evidence of cerebral compression such as a contralateral hemiparesis and homolateral dilatation of the pupil.

Eleven of the twelve patients were unconscious following the trauma. The twelfth patient was struck in the forehead by a back-firing crank handle without immediate loss of consciousness. The initial period of unconsciousness lasted from a few moments to as long as one to two hours. The more severe the injury, the longer the period of immediate unconsciousness. The severity of the syndrome of extradural hemorrhage was in no way related to the duration of the initial unconsciousness.

All patients had a lucid interval, the length of which in two instances was obscured by severe alcoholic intoxication. In the six patients whose course was very rapid and in whom a middle meningeal

main stem vessel was found to be the bleeding point the lucid interval was consistently about one hour and the coma which followed was rapidly progressive being complete at two to two and one-half hours after the injury. In the six patients whose course was relatively slow and in whom a smaller branch of the middle meningeal vessel was found to be the origin of hemorrhage the lucid interval was generally

TABLE I

Case No.	Age and Sex	Severity of Injury	Initial Unconsciousness	Lucid Interval	Focal Signs	Onset of Coma	Onset of Decerebration	Interval to Operation	Mid. Meningeal Vessel Involved	Result
1	32, M	Minor	Acute alcoholism	Acute alcoholism	?	?	?	Not operated	Principal vessel	Death on arrival at hospital 19 hr. after injury
2	21, M	Severe	10 min.	1 hr.	Ipsolat. dilated pupil and contralat hemiparesis	2 hr.	0	3½ hr.	Principal vessel	Recovered
3	46, M	Minor	Few sec.	1 hr.	Ipsolat. dilated pupil and contralat hemiparesis	2 hr.	Marked at 6 hr.	8 hr.	Principal vessel	Died in 24 hr.
4	7, M	Minor	Few sec.	1 hr.	Ipsolat. dilated pupil and contralat hemiparesis	2 hr.	Bilateral Babinski at 3 hr.	3½ hr.	Principal vessel	Recovered
5	33, M	Minor	Few sec. (alcoholism)	?	Ipsolat. dilated pupil and contralat hemiparesis	5 hr. maximum	Marked at 5½ hr.	8½ hr.	Principal vessel	Died in 36 hr.
6	9, M	Severe	30 min.	30 min.	Ipsolat. dilated pupil and contralat hemiparesis	2½ hr.	Bilateral Babinski at 2½ hr.	3 hr.	Principal vessel	Died in 72 hr.
7	17, M	Minor	0	24 hr.	0	0	0	72 hr.	Small anterior branch	Recovered
8	53, M	Moderate	5 min.	38 hr.	Contralat hemiparesis	0	0	50 hr.	Small anterior branch	Recovered
9	31, M	Severe	1 hr.	2 hr.	Ipsolat. dilated pupil and contralat hemiparesis	0	0	30 hr.	Small posterior branch	Recovered
10	31, M	Severe	15 to 30 min.	3 hr.	Ipsolat. dilated pupil and contralat hemiparesis	0	0	22 hr.	Small posterior branch	Recovered
11	22, M	Severe	2 hr.	3 hr.	Dilated ipsolat. pupil	0	0	Not operated	Small posterior branch	Died—abruptly 10 days after injury
12	10, M	Minor	5 to 10 min.	1 hr.	Dilated ipsilat. pupil	Semi-comat. at 6 hr.	Bilateral Babinski at 5½ hr.	8 hr.	Moderate sized posterior branch	Recovered

longer, one, two, three, three, twenty-four and thirty-eight hours, respectively. The two patients with prolonged intervals of lucidity were both instances of extradural hemorrhage from a relatively small anterior (frontal) branch of the middle meningeal vessel. All four patients in this group with more slowly progressive extradural hemorrhage but who had lucid intervals of only one to three hours had the hemorrhage occurring from a small posterior branch. However, in these patients the succeeding period of altered consciousness was only slowly progressive and complete coma never induced even as long as several days after the injury.

We do not wish to overstress the importance of the lucid interval by its presence in eleven of our twelve cases. We fully believe, as has been stated by Gurdjian and Webster,[1] that in severely injured individuals the deepening of an already unconscious state would be even more commonly observed than the lucid interval. However, in instances of relatively minor injuries as was the situation in many of our patients, one would expect the presence of a lucid interval to be invariable.

All of our patients with bleeding from the principal middle meningeal vessel had evidences of a contralateral hemiparesis and an ipsilateral dilated pupil when first seen by the surgeon. Neither of the two patients with hemorrhage from a frontal branch of the middle meningeal vessel had pupillary changes. On the other hand all four patients with a posterior branch middle meningeal hemorrhage had an ipsilateral dilated pupil and only two of these had a contralateral hemiparesis.

We wish to emphasize an interesting group of signs noted in some of our patients. These were in our opinion evidences of decerebrate rigidity: rigidity of all four limbs and neck with evidence of bilateral pyramidal tract involvement as indicated by the Babinski reflex and the presence of ankle clonus. Two patients definitely presented these signs and one of these even had "tonic fits" just prior to operation.

Both were instances of main trunk middle meningeal arterial bleeding and both died in spite of successful evacuation of the hemorrhage at eight and eight and one-half hours, respectively, after injury. Three other patients had bilateral Babinski reflexes but no rigidity of the ipsilateral extremities. One was a deeply comatose child of seven, operated upon successfully three hours after injury and the main trunk of the middle meningeal vessel identified as the bleeding point. A second was a patient with hemorrhage from a large size posterior branch of the middle meningeal vessel in coma at the end of eight hours but nevertheless saved by operation. The third was a severely injured child in whom a large hemorrhage from the main middle meningeal vessel was evacuated three hours after injury. Unfortunately, death occurred abruptly forty-eight hours later from increased intracranial pressure and medullary compression following a lumbar puncture.

The cause of decerebrate symptoms is the herniation of the uncus of the temporal lobe through the incisura of the tentorium with pressure upon the mid-brain. Severe compression of the mid-brain undoubtedly causes hemorrhage into this vital area and the damage is now irreversible. That this occurs most prominently in temporal lobe mass lesions has been frequently and recently emphasized.[2] Extradural hemorrhage, particularly when resulting from rupture of the main vessel near its point of entrance into the cranium, exerts pressure principally upon the temporal lobe. The homolateral dilated pupil apparently results from this same mechanism, the third cranial nerve being exposed to pressure from the temporal uncus as it passes forward through the incisura of the tentorium.

The vital signs reflected the increasing cerebral compression. This was particularly true of the pulse which in the rapidly progressing cases invariably was below 60 per minute and frequently below 50. The respirations were not as consistently altered as the pulse and early were increased as often as slowed; although in those pa-

tients who died, the respirations were always rapid, deep and stertorous. The pulse pressure was generally increased due to an increase in systolic pressure. The increase in pulse pressure was generally of the magnitude of 20 to 40 mm. of mercury. The temperature early in the clinical course was elevated possibly one or two degrees, but terminally all moribund patients had a hyperthermia. The hyperthermia accompanied or soon followed the appearance of decerebration.

TREATMENT AND RESULTS

The only treatment is prompt evacuation of the hemorrhage. A subtemporal decompression is generally agreed to be the approach of choice: (1) It affords adequate exposure for evacuation of the clot and for control of the bleeding point. (2) It is a procedure rapidly accomplished with minimal operating room preparation. (3) It provides a decompression for postoperative cerebral edema and increased intracranial pressure.

The dura should be opened and the underlying cortex inspected. The presence of a subdural collection of spinal fluid, either clear or blood-tinged, beneath the area of extradural hemorrhage is not unusual. This was noted in four of our twelve cases and has been remarked upon by other observers.[1] Though the origin and significance of this subdural collection is not clear, it is often large enough to require evacuation. Other points in favor of opening the dura are that it provides better decompression for postoperative cerebral edema and makes possible direct observation of any cerebral contusion. Extensive cortical damage has therapeutic as well as prognostic significance. On rare occasions a subdural collection of blood may accompany an extradural hemorrhage.

Our series has led us to revise our opinions as to the rapidity with which the syndrome of extradural hemorrhage can progress and how truly prompt surgical intervention must be. In instances of rupture of the main stem or principal branch of the middle meningeal artery surgical intervention cannot hope to be successful unless instituted within three to six hours after the injury. Shortly after the third hour following the rupture of the principal middle meningeal vessel decerebration occurs. Thereafter it is probable the neurologic damage caused by the pressure of the hemorrhage is irreversible. We have had six patients in this category and only two survived. Both patients that survived were operated upon within less than three and one-half hours after their injury.

When only a small branch is the source of the hemorrhage, the surgeon is afforded considerably more time in which operation can be successful. In our series we had two patients the source of whose bleeding was a small anterior branch. In both the lucid interval had been prolonged (twenty-four and forty-eight hours) and operation was successful fifty and seventy-two hours after injury. In four patients the small vessel that had been opened lay posteriorly in the cranium. The lucid interval was short (one to three hours) but the succeeding period of altered consciousness was only slowly progressive and coma never occurred. Three were operated upon successfully eight to thirty hours after injury. The fourth of this group died unoperated upon owing to an error in diagnosis. The patient had never really become more than stuporous and his exodus occurred abruptly ten days after the injury.

In the entire series the operative mortality was 30 per cent, two deaths being instances of late operation upon main stem middle meningeal bleeding and the third operative fatality occurred as a result of severe associated brain damage. The case mortality was 42 per cent, there being two additional patients dying without operation. One of the nonoperative deaths has been discussed above as being due to an error in diagnosis and the other was an instance of bleeding from the principal middle meningeal vessel in a patient who was dead upon arrival at the hospital seventeen hours after injury. This mortal-

ity is in the range reported in recent years: Munro[3] reports an overall mortality of 55 per cent, Kennedy and Wortis[4] 72 per cent, McKenzie[5] 45 per cent and Gurdjian and Webster[1] 27 per cent.

SUMMARY AND CONCLUSIONS

1. The urgency for surgical treatment in cases of intracranial extradural hemorrhage is dependent upon the portion of the middle meningeal complex that is the origin of the bleeding and independent of the severity of the initial trauma.

2. When the origin of the bleeding is the principal middle meningeal vessel, the lucid interval is almost invariably one hour or less and the succeeding coma complete at two hours following the injury. Shortly after the third hour following trauma evidences of decerebration become manifest, which when full blown indicate that the neurologic damage to the patient is irreversible. Therefore, in instances of hemorrhage from the principal middle meningeal vessel operation must be undertaken within three to six hours of the injury if the patient is to be saved.

3. Bleeding from an anterior branch of the middle meningeal vessel is associated with a prolonged lucid interval of one to two days. The succeeding alteration of consciousness is not severe and only slowly progressive. Pupillary changes are not the rule though a contralateral hemiparesis may be present.

4. In instances of extradural hemorrhage from a posterior branch of the middle meningeal artery the lucid interval is generally of one to three hours' duration, but the succeeding state of alteration of consciousness is not profound and only slowly progressive. Ipsilateral dilatation of the pupil appears to be constant. A contralateral hemiparesis was present in only half of our cases of extradural hemorrhage originating from a posterior middle meningeal branch.

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MUCOCELE OF THE APPENDIX*

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MUCOCELE of the appendix, also known as pseudomucinous cyst or mucoid disease of the appendix, is a condition of a comparatively rare occurrence. Woodruff and McDonald[1] found 146 cases of cyst of the appendix in 43,000 appendectomies, an incidence of 0.3 per cent, at the Mayo Clinic. Similar tumors may involve one or both ovaries. When rupture of a mucocele of the appendix or ovary occurs, pseudomyxoma peritonei may follow although it is now known that it need not necessarily be a sequel of rupture in every case.

The condition was described in the appendix by Rokitansky in 1842 and by Virchow in 1863. Grodinsky and Rubnitz[2] produced it experimentally in rabbits by ligation of the base of the appendix after washing out the lumen and preserving the blood supply to the appendix at the time of ligation. Their work gives some clue as to the reason for the comparative rarity of the condition. It has been well demonstrated by Wangensteen and others that obstruction of the appendical lumen in the presence of pathogenic organisms produces acute appendicitis with perforation unless surgical operation intervenes. Apparently, in a few instances chronic localized obstruction of the appendix may occur in man without the presence of pathogenic bacteria beyond the point of obstruction, and in these few cases cystic distention of the distal lumen may occur.

The content of a mucocele is characteristically a jelly-like clear mucus. The mucus is said to be secreted by columnar cells lining the tubular glands of the mucous membrane. If secretion of mucus persists sufficiently, a cyst of the appendix may reach surprisingly large dimensions.

Bunch[3] states that the size may approach that of a gravid uterus at term. In some chronic cases, calcium deposited in the cyst may cause sufficient deposition of radioopaque salts to warrant the diagnosis of stone by roentgen examination.

Rupture of a pseudomucinous cyst is not followed by the typical picture of peritonitis, inasmuch as infecting organisms are usually not present within the cyst. Rupture of a mucocele may, however, produce ascites because of blockage of lymphatics by the gelatinous contents of the cyst. It has already been observed that not all cases of rupture of a mucocele are followed by pseudomyxoma peritonei. Woodruff and McDonald believe that pseudomyxoma peritonei occurs in those patients in whom the formation of a mucocele is associated with grade 1 adenocarcinoma and that the mucus which may fill the peritoneal cavity in these cases is secreted by malignant cells which have escaped from the original cyst and become implanted at various points on the peritoneal cavity. They found adenocarcinoma grade 1 on microscopic examination of ten of the 146 cystic appendices studied.

The following is a case report of a case of pseudomucinous cyst of the appendix complicated by the presence of an acute gangrenous cholecystitis:

CASE REPORTS

CASE 1. The patient was a white male, age seventy-six. On admission he stated that he had pain in the abdomen for three days prior to admission. The pain was crampy in character and was accompanied by vomiting. After the onset of pain it became localized on the right side of the abdomen, particularly in the right upper quadrant. The bowels had not moved since the onset of this illness. The past medical

FIG. 1. Case I. A, acute gangrenous gallbladder; B, mucocele of the appendix showing a portion of the gelatinous contents.

history was insignificant except for a milder attack of pain and vomiting one year ago similar to the present attack. A physical examination revealed a slightly distended abdomen with very marked tenderness and a muscle spasm on the right side which was most severe in the right upper quadrant. No abdominal mass or tumor could be felt because of the extreme tenderness and muscle splinting. Although the admitting diagnosis was acute appendicitis, it was our belief that symptoms were probably due to acute cholecystitis. Temperature shortly after admission rose to 101.2°F. and gradually subsided so that operation was performed on the day after admission. The white blood count on the day of admission was 21,450, with 85 per cent polymorphonuclear cells.

On May 18, 1946, operation was performed under spinal anesthesia. On opening the abdomen through a lower right pararectus incision, a cystic mass the size of a grapefruit was found. This proved to be a mucocele of the appendix which had recently perforated, discharging some of the mucoid contents into the peritoneal cavity. Some of the small bowel in the region of the cyst showed a furry thickening of the serous coat where it had come in contact with the contents of the cyst. A culture of the cystic contents revealed no growth in seventy-two hours and no organisms were seen on direct smear. All the mucus that was in the peritoneal cavity was removed as completely as possible. The cyst was removed after

clamping its attachment to the cecum, and the stump was closed with a continuous Connell suture of atraumatic chromic catgut reinforced by interrupted sutures of linen thread. Examination of the gallbladder then showed a very tense, thick-walled organ, so the incision was extended upward and an acute gangrenous gallbladder was removed. A Penrose drain was placed in the gallbladder bed but no drainage was used in the region of the mucocele. Convalescence was uneventful and the patient was discharged on the fourteenth postoperative day.

The pathologic report of the specimens (Fig. 1) by Dr. Helen Ingelby was as follows:

Macroscopically, 10 cc. of thick gelatinous yellow fluid with slight amount of blood was examined. The appendix consisted of a loculated cyst 12 by 10.5 by 5 cm. The smaller loculus was 6 by 4 cm. At the junction of the two was a papillary projection 1.2 by 1.5 cm. Two other small projections were found in the inner surface of the cyst. The cyst was filled with mucoid material. The cyst wall consisted of hyalinized connective tissue. What seemed to be the proximal portion of the appendix was found on one side of the larger cyst.

A piece of fat, apparently meso-appendix, was sent with the specimen. The fat appeared a little atrophic. The peritoneum over it was thickened.

The gallbladder had been opened. It measured 12 by 6 cm. The wall showed commencing gangrene throughout. Most of the mucosa had

been destroyed. In one place the wall appeared about to perforate. The outer wall of the gallbladder was roughened and showed purplish congestion with green discolored areas.

Microscopically, the projection into the appendix consisted of fibromuscular tissue covered by distorted glands. The glands contained enormous amounts of mucus. A few inflammatory cells were scattered through the wall. Some areas in the wall were undergoing mucoid degeneration.

A section of a small piece of mucosa near the cecum showed a few cecal glands and submucosal with a thickened muscular wall. The subperitoneal tissue presented many inflammatory cells and showed some hyaline and mucoid degeneration.

Cell block from the contents of the appendix showed inspissated mucin and a scattering of red cells.

The wall of the gallbladder was gangrenous throughout. Inflammatory cells were found in the surrounding fat and also an abscess.

The lymph node showed inflammatory hyperplasia of the reticulum with bile particles in the reticulum cells. Many of the germinal centers were necrotic.

Pathologic diagnosis: Enormous mucocele of appendix; gangrenous cholecystitis.

While in most instances the contents of a mucocele are sterile, a case rarely occurs in which infection is superimposed upon a mucocele. Such a case was encountered several years ago:

CASE II. The patient was an obese male, age sixty, complaining of pain in the right lower quadrant for twenty-four hours. Several hours after the onset of pain he took a dose of magnesium sulfate and following this, became nauseated but did not vomit. His family physician was called and sent him to the hospital with a diagnosis of acute appendicitis. On physical examination he was found to be a very obese man with a thick panniculus. The blood pressure was 130/80 and the heart was found to be enlarged. Examination of the abdomen revealed a marked degree of rigidity in the right lower quadrant. The white blood count on admission was 16,000 with 82 per cent polymorphonuclear cells. The urine specific gravity was 1.025 with a trace of albumin and 4 to 6 red blood cells per high powered field were noted. Fasting blood sugar was 154 mg.

per cent, and the blood and urea nitrogen was 26 mg. per cent.

An appendectomy was performed under spinal anesthesia on the day of admission and a greatly dilated appendix was removed. Grossly, the appendix was of the acute suppurative type, and there was much induration of the cecum at the base of the appendix so that the stump could not be inverted. The abdomen was closed without drainage. On the day following the operation rales were noted through both lungs with expectoration of bloody mucus. The patient was markedly cyanotic. Response to oxygen therapy and $\frac{1}{50}$ gr. of atropine sulfate hypodermically was poor and the patient died the following day. Autopsy revealed a very severe degree of myocardosis and it was noted that in the wall of the left ventricle the muscle was replaced by fatty tissue in many areas. There was no evidence of peritonitis.

The pathologic examination of the appendix was reported as follows: The appendix was 7 by 3½ by 3 cm. and was filled with gelatinous material. A section of tissue was lined with multiple layers of tall columnar epithelium. Some of these simulated goblet cells. The subepithelial zone contained a thin layer of circular involuntary muscle. The lumen was filled with a copious amount of mucoid muscle. Interspersed throughout were a large number of polymorphonuclear lymphatic and mononuclear cells. Involuntary muscle showed hyaline changes. Inflammatory exudate cells were arranged in bundles and were surrounded by areas of necrosis.

Diagnosis: Acute suppurative mucocele of the appendix.

A third case is interesting because it is somewhat different from the two previously noted and introduces another possible cause for the formation of mucocele:

CASE III. The patient was a man, age thirty-nine, admitted to the Jewish Hospital on March 22, 1935. Six months previously he had undergone drainage of an appendical abscess without removal of the appendix. He was advised to return at a later date for an appendectomy. On the day after his readmission the appendix was removed under spinal anesthesia. The postoperative course was uneventful.

Macroscopic examination of the appendix showed it to be 6 cm. in length by 3 cm. The

serosa was smooth and the muscularis slightly thickened. The mucosa was pale, and the lumen contained gelatinous material.

Microscopically, a section of the appendix showed marked dilatation of the lumen, which was lined with a single layer of columnar epithelium of the goblet variety. The nuclei were located at the base of the individual cells. The glands of Lieberkuhn were diminished in number. No distinct lymph follicles were seen. A small number of lymphocytes were observed in the mucosa but the muscularis and serosa were normal.

In this case it is possible that a mucocele was present at the time of the original attack of acute suppurative appendicitis with perforation and subsequent abscess formation. However, it seems more probable that the mucocele formed subsequent to the burning out of the infection in the appendix, and this may be cited as an additional source of mucocele formation.

SUMMARY AND CONCLUSION

1. Mucocele of the appendix is a relatively uncommon tumor. It may be asymptomatic for years. Our experience would indicate that infection may be superimposed upon a mucocele and give symptoms similar to those of acute appendicitis. Rupture of a mucocele may occur without acute infection as a result of overdistention by the mucus contents.

2. Theories as to origin of mucocele of the appendix have been reviewed.

3. Three cases of mucocele of the appendix encountered in the past ten years have been reviewed.

4. Pseudomyxoma peritonei was not observed as a complication following removal of the mucocele in any of these cases.

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GLOSSOPHARYNGEAL NERVE BLOCK*

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INTERRUPTION of the glossopharyngeal nerve by the deposition of local anesthetic solutions is useful in the accurate diagnosis of tic douloureux or neuralgia in which this nerve is involved and in providing anesthesia for operative intervention upon the posterior third of the tongue. This discussion will include a technic for completing glossopharyngeal nerve block safely and effectively, a résumé of the pertinent anatomy and descriptive case reports.

The most common method hitherto employed for anesthetizing the peripheral glossopharyngeal nerve supply was that of Labat.[1] It is accomplished by a fan-wise field block performed by inserting a needle just above the mid-point of the hyoid bone and injecting against the tip of the finger which is placed against the base of the tongue near the epiglottis. The maneuvers performed are time consuming, uncomfortable for the patient and fail to provide adequate anesthesia in most instances. A simpler and more efficacious interruption of the glossopharyngeal nerve pathways is desirable.

ANATOMY

The glossopharyngeal nerves are paired, mixed cranial nerves. Each is attached to the medulla by several roots which enter the posterolateral sulcus, dorsal to the anterior end of the olivary body. These filaments blend laterally into a trunk in front of the vagus. The nerve leaves the skull through the jugular foramen with the vagus and accessory nerves. It is enclosed, however, in its own sheath of dura mater. After emerging from the jugular foramen, the glossopharyngeal nerve descends between the internal carotid artery and the internal jugular vein to the lateral side of the vagus. It then passes medial to the styloid process and anteriorly to the lower border of the stylopharyngeus. (Fig. 1.) After winding around the latter it extends forward between the internal and external carotid arteries, following the upper border of the middle constrictor medial to the hyoglossus and enters the pharynx where it spreads out over the posterior third of the tongue in the submucosa.

The glossopharyngeal nerve supplies a motor branch to the stylopharyngeus and joins the pharyngeal plexus to which the vagus and sympathetics also contribute fibers to supply sensation to the pharyngeal walls, tonsils and soft palate and is thus implicated in the initiation of the swallowing reflex. It terminates in the posterior third of the tongue as fibers which subserve sensation and taste from this area. The other branches of the ninth nerve are not a direct concern of this discussion.

TECHNIC

The presence of constant deep and superficial landmarks, preferably bony prominences, aid in the accuracy and success of nerve blocking. The technic to be described includes reliable bony landmarks throughout. The external reference points are illustrated in Figure 2 and the precise locus of anesthetic interruption in Figure 1.

The patient is placed in the supine position and the head turned toward the opposite side to about 45 degrees with the sagittal plane of the body. The tip of the mastoid bone is then identified and marked with a skin pencil. The angle of the mandible is also located and marked. A line is drawn connecting these points and is then bisected. A skin wheal is raised at the mid-
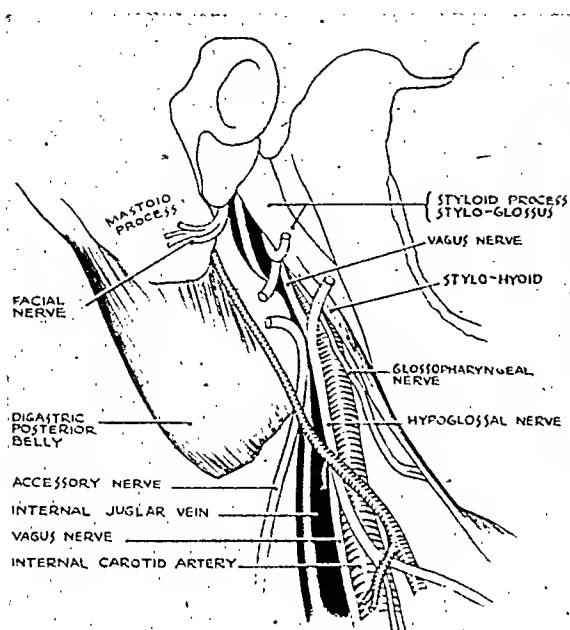
FIG. 1. A diagrammatical representation of the essential anatomic relationships of the glossopharyngeal nerve.

FIG. 2. The cross indicates the site of the skin wheal used for glossopharyngeal nerve block.

point of the line joining the mastoid tip and the angle of the mandible. Contact with the styloid process is effected by inserting a 5 cm. fine caliber needle through the wheal in a direction vertical to the skin. After the styloid process is encountered at a depth varying from 2 to 4 cm. medial to the skin surface, the needle is reinserted in a manner which permits the point to pass 0.5 cm. deeper and posterior to the bony styloid. The needle point then lies immediately adjacent to the glossopharyngeal nerve and injection is completed very slowly after careful aspiration. A total of 4 to 8 cc. of 1.5 per cent procaine solution on each side will provide adequate anesthesia of the posterior third of the tongue for approximately seventy-five minutes.

Although theoretically probable, concomitant block of the facial and vagus nerves has not been observed. Injection into a major blood vessel can be avoided uniformly by careful and repeated aspiration during the procedure.

The technic described has been successfully completed for anesthesia to facilitate surgical procedures upon the tongue and as a diagnostic procedure to select patients for intracranial ninth nerve section.

CASE REPORTS

CASE 1. G. R., sixty-one year old white male, was scheduled for insertion of radium into the posterior third of the tongue for the treatment of carcinoma of this region. The patient had advanced arteriosclerotic heart disease and had experienced congestive heart failure. There was a secondary anemia of 2.5 million red blood cells and an hemoglobin of 7 Gm. per cent. The planned surgical procedure was expected to require approximately sixty minutes of anesthesia.

Premedication consisted of pentobarbital 0.1 Gm., morphine 0.01 Gm. and atropine 0.0004 Gm. thirty minutes prior to anesthesia. Bilateral glossopharyngeal block was performed in the manner previously described. Operation was begun fifteen minutes later and continued for a total of seventy-five minutes with complete comfort to the patient and no significant change in his condition. The surgeon experienced no technical difficulties in his manipulations.

CASE II. H. B., was a sixty-five year old male, with leukoplakia of the tongue most marked on the right side. He experienced constant severe burning pain in the tongue, markedly aggravated by talking and chewing. There was evidence of a marked loss of weight recently due to inadequate food intake. There was difficulty in evaluating the presence or absence of pain on swallowing. The tongue was uniformly tender to touch.

Bilateral lingual block with 1.5 per cent procaine was performed as a diagnostic measure. Pain relief in the anterior portion of the tongue was immediate but symptoms in the posterior third were unchanged. It was noted at this time that swallowing was significantly painful. The right glossopharyngeal nerve was then blocked during the period of lingual analgesia and all symptoms were alleviated. Pain recurred in approximately three hours. Therefore, the patient was referred to the Neurosurgical Service for surgical interruption of the glossopharyngeal and trigeminal nerves. Intracranial section of these nerves was performed on the right side to secure prolonged symptomatic relief. Postoperative review for approximately two months found the patient free from tongue pain except for a small area on the left side. Appropriate anesthesia of the areas supplied by the right trigeminal and glossopharyngeal nerves was maintained. The patient was comfortable and had been able to ingest food without pain.

SUMMARY

1. A new technic for the completion of glossopharyngeal nerve block is described.

2. Case reports are presented which illustrate the use of glossopharyngeal block in anesthetizing the base of the tongue for operative intervention and in providing an accurate diagnostic measure in the selection of patients for surgical section of the ninth cranial nerve.

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CONGENITAL CHONDROSTERNAL DEPRESSION (FUNNEL CHEST) RELIEVED BY CHONDROSTERNOPLASTY

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THE abnormality of the thorax, known commonly as funnel chest, pectus excavatum, trichterbrust, chone-chondrosternon, etc., is an oval depression of the chondrosternal area of the thorax. It is most often noted in early infancy and it becomes more marked as the child reaches maturity. The depth of the depression varies from a moderate depression of the sternoxiphoid angle to a severe depression in which the posterior surface of the sternoxiphoid angle is in contact with the vertebral column. In this report only the severe form is under consideration. It is brought to the attention of the physician because of its cosmetic and psychic effect or its interference with cardiorespiratory function. Due to this variance in degree a study of the incidence of the deformity may vary greatly with the investigator. In a review of the literature by Ochsner and DeBakey[9] twenty-eight cases were found in a total of 46,705 persons examined, an incidence of 0.059 per cent. It further disclosed that males predominate in the ratio of 4 to 1.

The etiology of this deformity has been attributed to various compressing forces such as pressure by the chin, knee or elbow during intrauterine life, obstructive conditions of the respiratory tract or rickets. Apparently heredity plays a conspicuous part as many case reports have shown entire families with varying degrees of the condition. In twelve families with 121 members Nowak[8] found forty-nine (40.4 per cent) affected with this deformity. J. Bauhin,[1] in 1596, was the first to attribute the deformity to the pull of the diaphragm. In 1860 Woillez[12] stated that the retraction of the central diaphragmatic tendon was the most likely cause of the deformity. The operative findings of Brown,[2] Ochsner and DeBakey,[9] Sweet[11] and Lester[5] leave very little doubt that the deformity is produced by a congenital, short central tendon of the diaphragm. Brown believed that the disease was congenital and progressive. On the basis of this etiology he was the first to attempt to prevent the deformity by operative means in infants who manifest the early signs of chondrosternal depression.

A study of the various published papers reveals that there is a wide choice of names. All are descriptive of the outward appearance of the thorax but all have something lacking which inspires others to try another name. Funnel chest, pectus excavatum, trichterbrust and chone-chondrosternon may fit the deformity but do not explain the genesis of the disorder. Everyone describes it as an oval or funnel-shaped depression of the lower portion of the sternum and its adjacent cartilages. The weight of evidence now indicates that the condition begins with respiration at birth and results from a congenital developmental abnormality of the central anterior portion of the diaphragm. It is with a great deal of hesitation that the author wishes to suggest a new name for this condition. However, in its simplicity and description, the term congenital chondrosternal depression seems to fit more adequately. In addition it can be classified apart from the acquired type which may result from a traumatic injury or disease.

The pathology which has been adequately described consists, in the main, of the typical deformity of the anterior chest wall the compression or displacement of the heart in the majority of cases into the left hemithorax and the compression of the

lungs, both by the deformity and the paradoxical respiration. This paradoxical respiration is the result of the inward pulling action of the shortened tendon on its attachment to the sternoxiphoid junction. The shape of the chest conforms to the abnormal action of the diaphragm. The literature lacks an adequate description of the pathologic picture since there are no autopsy reports. Only the operative reports indicate a short central tendon and a dense contracted substernal ligament. Hutcheson[4] in a study of six patients in whom there was a definite displacement of the heart to the left, observed that even a marked displacement is compatible with good cardiac function and a complete absence of chest symptoms.

The clinical picture varies from no discomfort to symptoms of total invalidism. The vast majority suffer no ill effects from the deformity and lead normal full active lives. Many are never aware of the condition until it is brought to their attention at routine examination. Manning[6] reports a severe case of congenital chondrosternal depression in a twenty-six year old airman who was an athlete and boxer and had a vital capacity of 5,300 cc. A comparatively small proportion suffer symptoms of cardiorespiratory embarrassment because of the compression upon the heart and great vessels. Brown[2] is of the opinion that the greatest cardiac embarrassment appears to be present when the heart is fixed in the midline and is compressed by the sternum.

In young children there is usually no psychologic effect, but in adolescents and young women the deformity is often emotionally disturbing to the point of warranting cosmetic plastic repair. Symptoms seldom appear in children before the age of five at which time they usually begin the period of greater physical activity. The most common complaints are fatigue, dyspnea on exertion, palpitation and dull precordial pain. These children often avoid participating in the more active sports and tend to become shy, retiring and asthenic. Physical examination reveals the thoracic deformity, tachycardia and varying degrees of cyanosis on effort. The vital capacity is usually diminished, sometimes as much as 50 per cent. Electrocardiographic tracings in the majority of cases show no abnormality. Occasional cases show right axis deviation. On deep inspiration the lower anterior thorax retracts most markedly at the sternoxiphoid junction. Commonly a systolic murmur is heard over the mitral area so that many of these children are treated as cases of rheumatic heart disease.

According to Sweet[11] there are two cardinal indications for the surgical correction of this congenital deformity: (1) disturbance of cardiac function and (2) unfavorable effect on the emotional state of the patient. The success he had with surgical treatment in two sisters convinced him of the value of the procedure. It is Nissen's[7] opinion that operative treatment is indicated only when cardiorespiratory embarrassment is present.

Brown[2] describes two operative procedures in the treatment of congenital chondrosternal depression. The first is a comparatively simple operation in young infants who manifest the inspiratory retraction of the sternum and the early indications of the deformity. It consists of incision through the sternoxiphoid joint, bluntly separating the anterior diaphragmatic attachment from the substernal ligament. According to Brown this prophylactic procedure prevents the fixed bony depression which would result as the child grows older. The second procedure is reserved for the older group with the fixed bony depression who manifest cardiorespiratory distress. This consists in resecting bilaterally the deformed portions of the cartilages, transecting the sternum just below the manubrium, cutting across the substernal ligament and elevation and traction of the sternum.

The first prophylactic procedure was performed on two infants by Brown[2] and on three infants by Lester[5] with uniformly very satisfactory results. In the operative
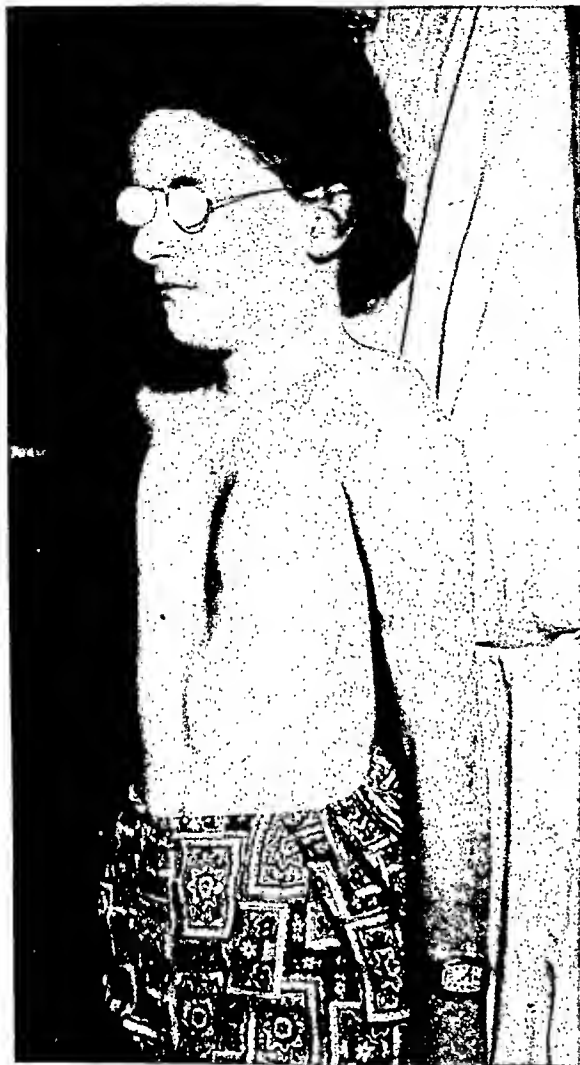
FIG. 1. Congenital chondrosternal depression. Preoperative picture taken in inspiration with shoulders held back.

treatment of the older group Ochsner and DeBakey,[9] in their very exhaustive review of the literature, discuss the three types of operations that have been performed for the correction of this deformity in thirty-one patients since 1911 when L. Meyer performed the first operation. They report the best results have been obtained in eight patients with sternal mobilization and traction after bilateral costochondral resection. Garnier[3] first performed this type of operation in 1934. Ochsner and DeBakey[9] report a very satisfactory result in their own case. In addition, between 1939 and 1946, American literature contains satisfactory results by this method in

three cases by Brown,[2] two cases by Sweet,[11] five cases by Lester[5] and one case by Phillips.[10] Nissen[7] reported satisfactory results in two patients after resection of the deformed portion of the sternum and suturing it horizontally to the costal cartilages in the reversed position.

The following case report is described to add another successful result by the operative procedure as modified by Brown:

CASE REPORT

C. R. age eleven years, a white female, was first examined in the Pediatric Clinic of Newark Beth Israel Hospital on October 9, 1939. Her mother stated that the child had been complaining of always being tired and upon the slightest exertion became faint. She did not play with other children or engage in any activity typical of her age group. She was referred to the cardiac clinic. On October 18th, when she came for an electrocardiograph, she appeared so ill that she was admitted to the hospital with a diagnosis of rheumatic heart disease.

Her father was living and well. He was of slight build with normal chest development. Her mother was living and well but had always been very much underweight. She was slightly round-shouldered and "flat-chested," but had no depression of the sternum. A younger sister of nine years was somewhat underweight but of normal development.

The patient's birth had been normal. She was breast fed for six weeks and then bottle fed. She appeared normally developed and began to walk and talk at the age of one. She had measles at the age of three, chicken pox at four, mumps at six and whooping cough at seven. She had a tonsillectomy at the age of three. Until February, 1939, according to the mother, she appeared in good health when she had a severe attack of "flu" and remained in bed for a whole month with high fever. From this time the mother stated that she noted her loss in weight and strength and the depression of the chest became more pronounced. Recently she had become nervous and complained of weakness and dyspnea on exertion. She also complained of precordial pain, frontal headaches and pains in muscles and joints of her legs.

Physical examination revealed the patient to be slender and bony. No dyspnea or cyanosis

were present. Her skin was pale, cool and moist. Her shoulders drooped forward and the chest was flattened. The sternum was depressed with the typical sharp angle at the junction of the gladiolus and the xiphoid. Breathing was abdominal in type and even on deep inspiration there was very little motion of the sternum. The depression measured 11 cm. long, 10 cm. wide and 5 cm. deep. The lungs were clear. The heart examination revealed a sinus arrhythmia and a systolic murmur over the apical area and at the base which was not transmitted. The point of maximum impulse was felt 3 cm. to the left of the nipple line in the fifth interspace. Blood pressure was 105/50. (Fig. 1.)

During her hospital stay the tuberculin test was negative. Venous pressure and circulation time were normal. Urine was negative and the blood count was normal except for a moderate secondary anemia. Roentgenograms showed displacement of the heart to the left and posteriorly. The heart appeared slightly enlarged with mitral configuration. Both central lung fields appeared congested with a slight degree of vascular accentuation. The asymmetry of the thoracic cage was noted with a considerable concavity of the lower three-fourths of the sternum causing a marked degree of narrowing of the ventrodorsal dimension of the thorax.

The electrocardiograph showed sinus arrhythmia, tachycardia, low voltage and right deviation of the electrical axis.

On October, 1939, the author in response to a consultation request, recommended a chondrosternoplastic operation to relieve the compression. This was not accepted because of the possible existence of rheumatic heart disease and the fact that surgical repair at this time was not well known. On November 6th the orthopedic service advised a special plaster jacket with a window over the sternal area in the hope that respiratory movements would correct the deformity. On November 16th a bilateral spica with arms in abduction was applied to the arms from the fingers to the chest and body down to the femoral trochanters. The patient was discharged on December 7, 1939, with the cast. The cast was worn for five months and removed in April but no improvement was noted.

The patient was readmitted to the Newark Beth Israel Hospital on June 10, 1940, for surgical treatment. At this admission a nota-

Fig. 2. Chondrosternoplasty with traction-elevation of sternum. Note silver wires at angle of Ludwig and lower end of gladiolus.

tion was made that the depression of the sternum was first noted when the child was about two years old and had steadily increased. Her chief complaints were precordial pain, dyspnea and faintness on exertion. The child showed no increase in growth but did gain about 10 pounds in weight since her first admission. She still appeared pale and undernourished. The patients vital capacity averaged 1,200 cc.

Operation was performed on June 11, 1940, under gas, oxygen, ether intratracheal anesthesia. A vertical mid-sternal incision from the angle of Ludwig to the end of the sternum was made. The skin and muscles were dissected off the costal cartilages on each side. Portions of depressed, angulated, costal cartilages were resected subperichondrially on each side from the second to the seventh costal cartilages. The xiphoid process was mobilized and removed. A narrow triangular wedge of the gladiolus of the sternum was removed transversely at the angle of Ludwig so that when the undersurface of the gladiolus was freed it could easily be elevated.

The dense fibrous tissue of the anterior mediastinum was incised transversely and the edges were seen to retract about 1 inch. The gladiolus was fixed in its new elevated position with two No. 20 silver wires. Another silver wire was taken in the lower end of the gladiolus and carried through the muscles and skin on each side 2 inches lateral to the incision. Muscle layers and fascia were sutured with interrupted No. 1 chromic sutures and the skin incision was sutured with interrupted silk. Flat rubber sponges were glued to the skin below the nipples on each side and a narrow curved wire bridge was placed across the lower chest with each end resting on the rubber sponges. The silver wire from the lower end of the sternum was tied on the wire bridge anchoring the raised sternum. Dressings were applied under the wire bridge covering the incision. The operation lasted two hours at the end of which time the condition of the patient remained good. At no time were the pleural cavities entered. (Fig. 2.)

The postoperative course was uneventful and the patient was discharged on the sixteenth postoperative day with the wire splint in place and the wound healed per primum. A shoulder brace was prescribed to correct the poor posture.

On discharge it was noted that the sternum was on a level with the rest of the anterior chest wall. The anteroposterior diameter of the chest was increased by 2 cm., now 14½ cm. Prior to operation the sternum was further depressed on inspiration but since operation it was elevated on inspiration. The heart murmur could no longer be heard. Very little change was noted in the electrocardiograms.

A follow-up interview about one year later revealed the fact that she indulged in normal activities, roller-skating and bicycling and joined in the play of her friends without any dyspnea or sensation of faintness. The symptomatic result was most satisfactory and a recent communication from the patient stated that she works steadily in a department store.

As far as the cosmetic correction of the deformity is concerned, the author agrees with Lester[5] that a 75 per cent correction should be considered a satisfactory result.

An inverted Y-shape incision instead of a straight mid-sternal incision provides a wider exposure of the lower costal cartilages.

SUMMARY AND CONCLUSIONS

1. A case of congenital chondrosternal depression which was successfully corrected surgically by a chondrosternoplasty is presented.

2. A more appropriate name for this thoracic deformity is suggested, namely, congenital chondrosternal depression.

3. This congenital deformity may exist to a severe degree without knowledge of the patient or the presence of any untoward effect.

4. In a relatively small proportion there may be symptoms of cardiorespiratory embarrassment as well as severe and harmful psychologic effects.

5. To these patients surgery, by means of a chondrosternoplasty, offers complete relief of symptoms as well as improvement in the outward deformity. The operative procedure as advocated by Brown will prevent the bony deformity if it is applied in infants who are affected.

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GASTRIC RESECTION FOR DUODENAL ULCER

SURGICAL TREATMENT AND FOLLOW-UP STUDY

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WITH the last decade the pendulum has swung to a more radical method of treatment in chronic duodenal ulcers. It was the opinion of surgeons before this era that less radical measures were advisable. Since adapting gastric resection as the usual procedure for chronic duodenal ulcers, an attempt was made to analyze sixty-two consecutive patients who had resection done during the period from January, 1939 to January, 1943.

These patients were all male; thirty-six were veterans of World War I, twenty-two were merchant seamen and four were coast guardsmen, three of the latter already were retired on disability. Of this entire group only three were negroes. Their occupation in the seamen group ranged from captain, engineer and purser down to deckhand and mess boy. In the veteran group their occupations varied from salesman, janitor, carpenter, welder, delivery man, studio decorator, shipping clerk, dispatcher, fireman, chauffeur and laborer.

INCIDENCE OF GASTRIC RESECTIONS FOR DUODENAL ULCER

	No.	Per Cent
Resection for duodenal ulcer	62	56.4
Resection for gastric ulcer	30	27.3
Resection for carcinoma	16	14.5
Resection for gastric colic fistula	2	1.8
Total	110	100

Indications. The principles of management of malignant lesions are clear; the chief problem is early recognition. The common indications for operative intervention in duodenal ulcer, aside from perforation, pyloric obstruction and persistent bleeding, are chronicity and sufficient severity of symptoms which cannot be controlled by adequate medical treatment. Even then the results will depend to a large extent on the skill with which patients are selected for operation. In addition age, sex, occupation and personality of the patient must be taken into consideration since all of these factors influence results in any form of treatment. While intractability is the first essential, there are occasional cases in which operative procedure may be indicated early in the disease.

INDICATIONS IN THIS SERIES

	No. Cases
Resection for pyloric obstruction	9
Resection for intractability	31
Resection for bleeding	22

It is interesting to note that ten of these patients had been operated upon for perforations and in one instance the patient had two previous operations for perforation. In addition, six of the patients had been operated upon, before undergoing gastro-enterostomy, and a marginal ulcer was found at the time of gastric resection in two of these patients. Two patients were operated upon for acute hemorrhage.

DURATION OF SYMPTOMS

	No. Cases
2–3 years	15
5–10 years	20
10–15 years	13
15–20 years	14

AGE

	No. Cases
20–30 years	3
30–40 years	10
40–50 years	30
50–60 years	19

Gastric Analysis. Since the rationale of radical treatment in chronic duodenal ulcer is removing the most active portion of the stomach along with the acid-stimulating portion of the gastric mucosa and acid-secreting cells, special attention was given to preoperative gastric analysis. However, in reviewing these cases no record of pre-operative gastric acidity could be found in twelve cases. Twenty-seven patients showed hyperacidity. Fasting stomach contents were used for the analysis before and after histamine. Gastric analysis was repeated between six months and a year postoperatively. In the twenty-seven cases listed as hyperacidity the records show a definite lowering of the acidity in twenty-two patients postoperatively. In one patient it remained the same. Of the six patients who complained of mild indigestion postoperatively, the free HCl and total acidity was low. No correlation or conclusion could be reached from this study in regard to gastric acidity. Low acidity was found in nine patients and normal acidity in fourteen.

Type of Resection. The Hofmeister modification of the Polya type of anastomosis was performed in all cases except five. The anastomosis was antecolic in all cases and the great majority were isoperistaltic. Fine chromic catgut was used as suture material, reinforcing the lesser and greater curvature angles with omental tags. Three layers of suture material were used. The Billroth two-type of operation was used in five patients. Four of these patients had a previous gastro-enterostomy with reactivation of the duodenal ulcer and bleeding in three cases. The lower part of the stomach was resected along with the ulcer after close inspection of the gastro-enterostomy stoma and stomach mucosa. The fifth subject had a large bleeding perforating ulcer into the head of the pancreas with marked inflammatory reaction which could not be resected. An exclusion type of operation was performed, resecting the lower stomach and ulcer at a second stage which was surprisingly easy to carry out. Partial resection (less than one-half) was carried out on twenty-four patients and subtotal resection (more than one-half) thirty-eight patients. The ulcer was not removed in two cases but the stomach and pylorus was resected with satisfactory results.

Physiology. In the case of duodenal ulcer we are dealing with a poorly understood derangement of normal physiology. Hyperacidity, tissue susceptibility, psychologic disorder and various other factors of temperament, environment and economics play a part. The rôle of antral hormone cells is also to be considered. One must also remember that these individuals have not been helped by other medical measures.

The chief gastric functions are digestive and motor. Disturbance in the latter function gives rise to symptoms in the great majority of gastric disorders. The motor function is amplified in the pyloric antrum and along the lesser curvature. For this reason lesions in these areas cause more symptoms. Physiologists have also shown us that there is a chemical stimulant present in the antral mucosal cells which excites gastric secretion and promotes motor activity. This property has not been demonstrated in the other mucosal cells; therefore, resection of the lower part of the stomach removes the most active part and the mucous membrane which produces an active hormone for acid secretion.

Technical Difficulties. Attention to details is important in subtotal gastric resection in order to prevent complications. The stomach is especially difficult to approach in heavy-set, stout individuals with large adipose deposits in the mesentery and omentum. It is also extremely vascular which occasions bleeding if careful attention is not paid to hemostasis. With reasonable care, hemorrhage can be controlled, the stomach resected and anastomosis performed satisfactorily. In doing this it is quite important to free the mesentery from the lesser curvature to allow proper closure

and prevent subsequent leakage. Technical difficulties may arise when making the anastomosis in extremely high resection for carcinoma or in handling the duodenal stump when resection is performed for duodenal ulcer. It is with the latter that we are particularly concerned in this discussion. Apparently nothing in the operation is more often responsible for failure than the improper closure of the duodenal stump.

Ulcers occurring in the duodenum, scar and distort that structure and, together with thickening of the wall, make closure of the stump extremely difficult at times. For this reason thorough visualization of the duodenum is practiced with partial mobilization to determine its mobility, relation of the common duct to the ulcer, and extent of involvement of the pancreas before beginning the resection. At this time a decision is made as to the method of handling the remaining duodenal stump. It is often surprising how much duodenal wall one has available for closure after mobilization by freeing the scar tissue. Even after this satisfactory closure depends upon how well one is able to free the posterior wall from its pancreatic attachments. With patience, good hemostasis and some persistence this step can usually be accomplished. In this series, when the duodenal stump was long enough, clamps were used in its removal and closure. When it was not long enough, it was left open, freed of its contents by suction, and closed with Connell catgut suture followed by carefully applied silk interrupted sutures.

Complications. Leakage of the duodenal stump was the main complication in this series and accounted for two deaths. Another patient with leakage from the duodenal stump which walled-off was treated by incision and drainage followed by constant suction by way of a well placed catheter in the wound. Postoperative hemorrhage was not encountered. The vessels in the stomach were tied with fine catgut at time of operation. Pulmonary complications were minor and credit is given to use of an oxygen tent along with CO_2 inhalations. Thrombophlebitis was not encountered. One patient developed a subphrenic abscess and parotitis along with hypoproteinemia. This individual had been operated on for acute massive hemorrhage. A large ulcer was found involving the head of the pancreas and extending down dangerously close to the ampulla. An exclusion type of operation was performed resecting the ulcer and duodenum later. This is the only case in the series in which a two-stage operation was performed. The subphrenic abscess was drained and the parotitis treated with x-ray. Shock was obviated by rendering adequate preoperative and postoperative care in regard to fluid balance as presented by the individual patient.

There were two patients with malfunctioning stoma who required jejunostomy. Their symptoms cleared up without further trouble within thirty days. Another patient had intestinal obstruction six months postoperatively.

Disruption of the wound did not occur in this series. This, at times, is a rather serious complication and may be just enough to cause death in a seriously ill patient. A high midline incision was used and the wound was closed with silk. The peritoneum was closed with fine No. o braided silk and the fascial structure with No. ooo braided silk. Interrupted sutures were used with properly spaced mattress sutures in the fascia in addition to the straight interrupted stitches. No retention sutures were used. Since this study, one hundred cases have been closed in this manner with only one partial disruption and postoperative hernia observed. We believe that the midline incision and closure with silk are the approach and procedure of choice, especially in resection for duodenal ulcer. Particular mention of the incision and closure is made because the results were much better than our previous experience with closure of high right rectus and high midline incision with catgut.

Mortality. In the resection for duodenal ulcer there were two deaths, both due to peritonitis from blow-out of the duodenal stump. In the entire series there were three deaths; one from an emergency operation for massive gastric hemorrhage. The fatality rate was 3.2 per cent.

POSTOPERATIVE COMPLICATIONS

	No. Cases	Results	Comment
Leakage from duodenal stump.	3	2 deaths	Incision and suction drainage with recovery in one case
Parotitis and hypoproteinemia......	1	Recovered	Abscess drained
Malfunction of stoma..........	2	Recovered	Jejunostomy
Intestinal obstruction.	1	Recovered	6 months postopertive

Follow-up. Patients were studied for varying lengths of time as listed below:

No. Cases	Time Studied
14	6 months
13	1 year
16	2 years
9	3 years
10	4 years

Upon discharge from the hospital, patients were given careful, written instructions with regard to diet, use of tobacco and alcoholic beverages. In this entire group only ten patients stayed on a diet. Of these ten, six had mild indigestion occasionally. It is interesting to note that the large majority continue smoking, some to excess, and at least 50 per cent take an occasional drink with some listed as indulging in alcoholic liquors to excess. The six months' follow-up was recorded as good in fourteen patients. Of the others, thirty-four patients were following their regular occupations; twelve were doing less strenuous work and two were not working. Of the last two patients the result was good in one, the other had x-ray findings of gastrojejunal ulcer which was in accord with his symptoms. The other patient, complicated by gastrojejunal ulcer, returned two years postoperatively. This individual had been free of symptoms, smoked excessively and used alcoholic beverages to excess. He was working daily. His first symptom was weakness and in a few hours vomiting of a large quantity of blood. After this he began to have pain and the bleeding persisted. A high resection was done, removing the ulcer. He was followed postoperatively for one year and remained free of symptoms.

SUMMARY

1. A study of sixty-two cases of gastric resection for duodenal ulcer has been presented.

2. A follow-up study of these cases, with regard to gastric analysis and case fatality, is given.

3. Some facts as to technical difficulties encountered and evidence of complications in this series are presented.

4. There were two cases of gastrojejunal ulcer, following the resection in this group, representing an incidence of 3.2 per cent.

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Case Reports

HEMANGIOENDOTHELIOMA OF THE SALIVARY GLAND*

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HEMANGIOENDOTHELIOMA arising in the salivary glands is a rare condition. A case of such a tumor in a young infant is presented.

The most common lateral neck tumor in the young infant is a congenital cystic hygroma. Hemangioendothelioma in such a location is unusual. Smith[2] says, "A hemangioendothelioma of this region has never been observed at the Children's Hospital (Boston), nor has it been diagnosed prior to the present case in any of the 40,000 children born at the Boston Lying-in Hospital."

CASE REPORT

T. L., a male infant, was first seen on May 31, 1947, at the age of six weeks with no abnormalities noted. On June 20, 1947, the parents noted swelling of the left cheek. Three days later this was seen by a pediatrician who believed that the infant had a parotitis. A course of penicillin was given and the mass became somewhat smaller. It soon became large again and a course of sulfonamides was given with practically no effect. On July 14, 1947, the patient was seen in surgical consultation at which time the mass seemed to arise posterior and inferior to the left parotid gland, and the possible diagnosis of cystic hygroma was considered. The mass was followed for six weeks and grew steadily until it was about 8 cm. in diameter. It felt cystic and did not transilluminate light. The mass seemed to lie in the subcutaneous tissues of the neck. (Figs. 1 and 2.)

On August 19, 1947, an operation for removal of the tumor was performed by one of the authors (A. S. J.) under ether-oxygen endotracheal anesthesia. There was a large, wine-colored tumor about 6 by 5 by 4 cm. involving the left side of the neck from the transverse processes of the sixth cervical vertebra, the carotid and jugular vessels and the anterior surface of the sternocleidomastoid muscle to the skin. The tumor lay adjacent to the submaxillary and parotid glands but grossly seemed to be distinct from them. Many blood vessels extended from the tumor into surrounding tissues. Two lymph nodes were found in the area of the tumor. The tumor was excised intact. (Figs. 3 and 4.)

The postoperative course was uneventful, except for development of a partial, transient facial paralysis. (Fig. 5.)

The following pathologic report by Dr. Etheldred L. Schafer gives the gross and microscopic appearance of the specimen removed at surgery: "The gross specimen was a soft but not fluctuant, well encapsulated, dark red ovoid mass, measuring 5.5 by 4.5 by 3.5 cm. The external surface was faintly bosselated. The capsule was thin and transparent. Cut surfaces were coarsely lobulated, dark red, and slightly bulging. A small amount of blood exuded from the surface. Attached at one pole was a separate ovoid mass, measuring 1.5 cm. in maximum diameter, which had pinkish gray cut surfaces and resembled a lymph node.

"Numerous sections from the mass showed a vascular tumor involving salivary gland tissue. Some of the gland was visible at the margins of the tumor, and isolated glands and ducts occurred throughout the tumor. Delicate fibrous connective tissue septa divided the tumor into lobules reminiscent of the normal pattern of the salivary gland. In some regions, variable-sized blood-containing spaces with only thin walls separating them were numerous. These spaces were lined by endothelial cells which in some instances were plump. An

FIGS. 1 and 2. Preoperative photographs of infant.

FIG. 3. Tumor demonstrated at operation.

FIG. 4. Specimen removed at operation.

occasional mitotic figure was noted among these cells. In other foci, compact masses of small cells occurred between the blood spaces. These cells had indistinct cell outlines. Their nuclei were round or irregularly oval and fairly uniform in size. The chromatin material was irregularly dispersed throughout the nucleus; none contained a nucleolus. Mitotic figures were more numerous in these compact foci, numbering as many as 4 to 5 per high power field. An occasional multinucleated cell was noted. The lymph node at the periphery of the tumor was normal. (Figs. 6 and 7.)

"The tumor belongs in that group of tumors known as angiomata, specifically to the hemangiomata. The following must be considered in making a diagnosis: hemangioma (capillary type), hemangioendothelioma, hemangiopericytoma, and hemangioendothelioblastoma. The exact nature of the tumor cells and their relationship to blood vessels could not be demonstrated without special stains.

FIG. 5. Infant one month postoperatively.

Sections stained for reticulin by the Laidlaw method revealed anastomosing blood vessel channels with neoplastic cells largely confined within their reticulin sheaths. Thus, they are neoplastic endothelial cells, and the tumor is considered a hemangioendothelioma. Stout[1] who saw the sections and made the Laidlaw stains concurred in this diagnosis. Several other pathologists who viewed the sections variously diagnosed the tumor as a simple hemangioma or a hemangioendothelioma. The extreme cellularity of the tumor, the mitotic figures, and the multinucleated forms within the neoplastic cells remove this tumor from the group of simple capillary hemangioma. The presence of mitoses is thought to be consistent with a period of rapid growth of the tumor in an infant and not to indicate a malignant neoplasm.

"The diagnosis is a hemangioendothelioma arising in the salivary gland."

Lateral tumors of the neck are diagnostic and therapeutic problems met occasionally by head and neck surgeons. Along with cystic hygroma, other conditions to be considered in the young infant are infectious parotitis, tuberculous adenitis, acute

FIG. 6. Photomicrograph of a portion of the tumor to show presence of salivary gland ducts. Note the irregularly shaped blood-containing spaces separated by masses of neoplastic cells ($\times$ 150).
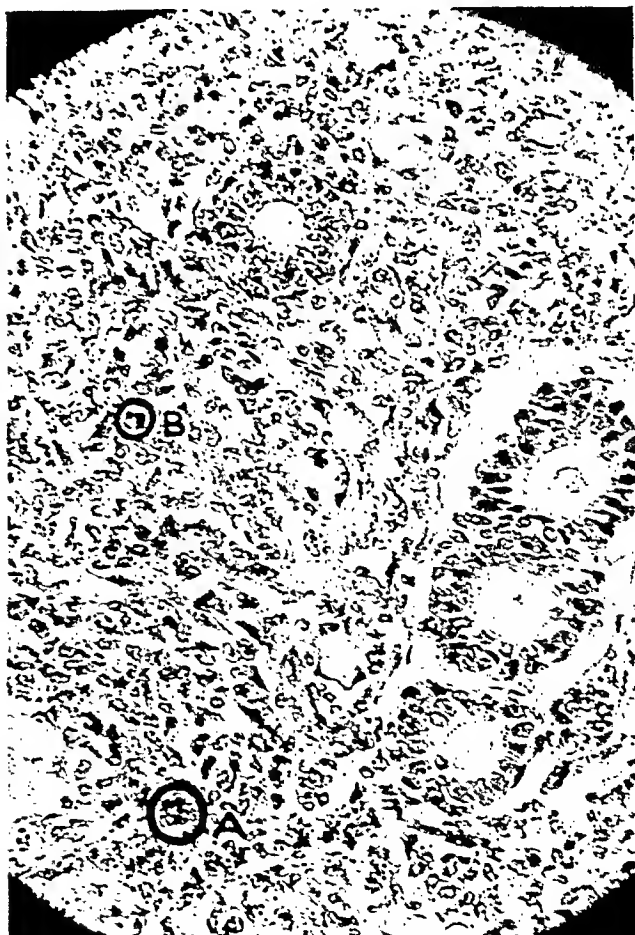
FIG. 7. Photomicrograph showing a portion of Figure 6 under higher magnification. Details of the neoplastic cells are more clearly shown. Note the multinucleated cell at A and the mitotic figure at B ($\times$ 450).

leukemia and branchiogenic cysts which rarely occur at this age.

In a review of American literature from 1916 no mention of this type of tumor arising in the salivary glands was found. Other tumors in this region are also rare. The sections from this case have been reviewed by a number of pathologists, all of whom concurred in the opinion that this was a benign vascular tumor; however, it was variously designated as a hemangioma (simple type) and a hemangioendothelioma. Three of the above pathologists mentioned had seen similar tumors, apparently primary, in a salivary gland. No reports have appeared in the literature.

Reading on this subject is difficult because the criteria for the classification of blood vessel tumors has not been clearly defined. Each author who has written on the subject has his own classification and terminology. Ewing,[3] Geschickter, Watson and McCarthy[4] and Stout[5] are among those who have classified these tumors.

From a surgeon's standpoint the pathologic interpretation of the tumor is important primarily from a prognostic aspect. Diagnosis in this case rests upon specific criteria set up by Stout[5] which are: "No tumor should be considered an hemangioendothelioma unless the following two features are present: first, the formation of atypical, endothelial cells in greater numbers than are required to line the vessels with a simple endothelial membrane; and, secondly, the formation of vascular tubes with a delicate framework of reticulin fibers and a marked tendency for their lumens to anastomose." Stout considers this a malignant tumor except in infants when it is usually benign.

Hemangioendotheliomas in the neck of infants have been reported by Smith,[2] Watson and McCarthy[4] and Sweitzer and Winer.[6] None of the three cases reported arose in salivary tissue. The tumors in the case reports of Watson and McCarthy and of Sweitzer and Winer were cutaneous and red.

SUMMARY

A case of benign hemangioendothelioma of the salivary glands is presented with a discussion of the gross and microscopic features involved and the possible benignancy of the tumor. A critical review of the literature reveals that these tumors are relatively infrequent.

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STENOSIS OF THE INTESTINE AFTER STRANGULATED HERNIA*

WITH FATAL COMPLICATION FOLLOWING INTESTINAL INTUBATION

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STENOSIS of the intestine following the reduction of a strangulated hernia is relatively uncommon. The first authentic record of such a complication is the case reported by Guignard in 1864. Cotte and Leriche, in 1905, collected thirty-nine cases and more recently Costa, Barry and Raw have added to the literature several instances of this sequela of strangulation.

We have recently observed at the Lahey Clinic two cases of delayed stricture and one of potential stenosis of the small intestine secondary to strangulated hernias. One patient presented, in addition, an unusual and fatal complication of intestinal intubation.

CASE REPORTS

CASE 1. A woman, fifty-two years of age, was first seen in the clinic on April 9, 1945, complaining of abdominal cramps and distention of two and one-half years' duration. In October, 1942, she had a strangulated femoral hernia repaired without resection of the involved intestine. The immediate postoperative course was characterized by diarrhea and abdominal cramps. Following discharge from the hospital, she had recurrent attacks of colicky pain which varied in frequency from once a week to once a month. The pain was accompanied by distention of the abdomen and anorexia. The weight loss was gradual. In February, 1945, she had several severe attacks during which vomiting was extreme.

Examination revealed that the patient was poorly nourished and chronically ill. Numerous loops of thickened, dilated intestine could be seen and felt through the thin anterior abdominal wall. Peristalsis was active.

Serial films of the small intestine after a barium meal showed several loops of moderately and greatly distended small bowel. The degree of dilatation was greatest in the lower ileum. Diagnosis was made of obstruction of the small bowel. Attempts to decompress the bowel with the Miller-Abbott tube were not successful and operation was advised.

The abdomen was opened on May 22, 1945. Numerous loops of dilated, thickened small bowel were encountered, extending from the upper jejunum to the lower ileum. Approximately 45 cm. (18 inches) proximal to the ileocecal valve there was an annular constriction of the intestine measuring about 2.5 cm. in length. The bowel was considerably scarred at this point. Below this stenotic area the intestine was collapsed. The proximal distended bowel was decompressed by gradually advancing the Miller-Abbott tube along the lumen of the intestine while continuous suction was exerted from above. An entero-enterostomy was performed by-passing the area of constriction.

Following operation, the temperature rose gradually and then assumed spiking, septic proportions. Abdominal distention did not subside. On the second postoperative day a sausage-shaped mass was palpable to the left of the incision. A moderate amount of blood drained through the Miller-Abbott tube but intestinal decompression was not accomplished. Despite vigorous supportive measures the patient failed to respond and died on the ninth postoperative day.

At autopsy the peritoneal cavity contained 2,000 cc. of foul-smelling fluid and considerable gas. The loops of intestine were matted together and greatly distended. Approximately 50 cm. distal to the ligament of Treitz there was a perforation of the jejunum. Just below the point of perforation 30 cm. of bowel was intussuscepted into a 10 cm. segment of intestine. Twenty cm. distal to this involvement there was another firm mass which represented

another area of intussusception. The entero-enterostomy in the terminal ileum was intact and widely patent.

CASE II. A woman, sixty-six years of age, was examined in the clinic on February 26, 1946. She complained of recurrent bouts of abdominal colic, associated with nausea and vomiting. Constipation which had been chronic for years, had become progressively worse.

In June, 1945, a strangulated hernia in the right inguinal area was repaired surgically without intestinal resection. The patient had considerable nausea and vomiting attended by crampy abdominal distress immediately after operation. These symptoms persisted and led to a second operation in September, 1945, ostensibly for the release of intra-abdominal adhesions. This procedure was followed by recurrent episodes of nausea, vomiting and abdominal pain. Gurgling abdominal noises and palpable abdominal masses had been noted by the patient. She had lost 34 pounds.

Physical examination revealed that the patient was thin and emaciated; she weighed 98 pounds. There was some mild arthritic deformity of the fingers. The heart was slightly enlarged but the sounds were clear. The abdominal wall was soft and flabby and large loops of hypertrophied small bowel were palpable throughout the abdomen. There were two well healed scars in the right lower quadrant of the abdomen. Diagnosis was made of small bowel obstruction, secondary to a contraction ring in an area of previous strangulation.

Following adequate decompression of the bowel with the aid of the Miller-Abbott tube, laparotomy was performed on February 28, 1946. The abdomen was entered through a low, right rectus incision. The proximal portion of the small intestine was dilated and the wall was thickened and edematous. There was an area of fibrosis and stenosis in the mid-ileum which almost occluded the lumen of the bowel. The strictured area measured about 5 cm. (2 inches) in length and was characterized by a densely scarred ring surrounding the bowel at each extremity of the area of narrowing. An entero-enterostomy was made so as to by-pass the stricture. The abdomen was closed in layers.

The postoperative course was uneventful and the patient was discharged from the hospital on the thirteenth day following operation. One month later she was entirely well and had gained 28 pounds.

CASE III. A man, sixty-two years of age, came to the clinic on March 22, 1946, complaining of pain in the abdomen of six months' duration. Two years before this visit he had had an operation elsewhere for recurrent pain in the right upper quadrant of the abdomen. He did not know what was found or what procedure was performed. He was well for eighteen months following operation, but then abdominal colic, increasing constipation and weight loss recurred. Upon two occasions during the six weeks before his examination at the clinic he had vomited a large amount of greenish fluid approximately six hours after eating.

The patient was extremely obese, with flabby musculature. The abdomen was pendulous and soft. There was mild tenderness in the region of the scar in the right upper quadrant of the abdomen where a defect was palpable, permitting protrusion of viscera upon coughing.

Routine laboratory data were normal. Cholecystograms revealed no gallbladder shadow. Barium studies of the stomach, duodenum and colon were negative.

He was placed on a reduction diet and bowel management which relieved his distress temporarily.

On September 16, 1946, following a large meal containing much roughage, the patient had severe abdominal pain, vomiting and obstipation. On examination a tense, tender, irreducible mass was found in the lower angle of the upper abdominal scar. He suddenly collapsed while in transit from one department of the clinic to another but revived spontaneously without residual symptoms. Diagnosis was made of strangulated hernia and operation was undertaken immediately. The sac was exposed and opened. A loop of small bowel about 20 cm. in length was liberated. It was a dusky purplish color with plastic exudate on the surface. One end of the confined loop was firmly adherent to the margin of the defect in the abdominal wall and the bowel at this point was narrowed and deeply creased. The color of the intestine returned to normal but the deep furrow did not relax. Consequently, a lateral entero-enterostomy was fashioned in order to circumvent the area of potential stricture. The hernial defect was repaired.

Recovery was devoid of incident and the patient was discharged from the hospital on the eleventh postoperative day. He has had no recurrence of abdominal distress.

COMMENTS

Pathology. Alterations in the bowel wall following strangulation vary from transient edema to gangrenous necrosis and perforation. Recovery of the involved intestine may be complete or partial. The mucosa is most seriously affected by strangulation and it may suffer from venous congestion, thrombosis and necrosis. The other coats of the intestine will be damaged, but to a lesser degree. Healing results in scarring if thrombosis and necrosis are marked.

The factors which determine the extent of damage to the bowel wall, as pointed out by Barry, are: (1) duration of strangulation, (2) tightness of the neck of the constricting sac and (3) resiliency of the vascular tree.

The stricture may be "physiologic" and transient because of interference with intestinal motility as a result of disturbance in the neuromuscular mechanism. Organic stricture, on the other hand, develops more gradually and persists until relieved by surgical measures. Organic stenosis may be annular or tubular in character. The former tends to involve a small segment of the bowel at either or both extremities of the constricted loop. Tubular involvement is longer and apparently results from impairment of the blood supply to the entire loop of strangulated bowel.

The most common location of these strictures is in the ileum. None have been reported in the large bowel. The intestine above the area of obstruction is dilated and thickened while that below the stenosis is deflated, thin and atrophied.

Delayed stenosis is more common following strangulated inguinal hernia than following femoral hernia although strangulation is more frequent in the latter type. Of thirty-seven cases collected by Jaboulay and Patel, twenty-seven patients had

inguinal, eight femoral and two umbilical hernias.

Symptoms. Symptoms may occur immediately after successful taxis or operative reduction of a strangulated hernia. The most common history is one of immediate postoperative obstruction which subsides for a few days or weeks and then recurs in the form of subacute or chronic obstruction. Recurrent colicky pain, nausea, vomiting, loss of weight and anemia constitute the classical clinical features. The immediate postoperative period is frequently characterized by an episode of bloody diarrhea which may indicate necrosis of the mucous membrane at the site of strangulation.

Physical signs include wasting, abdominal distention, palpable loops of distended, hypertrophied bowel and ausculatory evidence of excessive peristaltic activity. Serial films of the small bowel may or may not demonstrate slowing of the intestinal stream or narrowing of the lumen of the bowel.

Diagnosis should be made with reasonable accuracy in an individual with a past history of a strangulated hernia in whom the immediate postoperative course was marked by bloody diarrhea, associated with obstructive phenomena which subsided temporarily, only to recur in a more persistent fashion.

Treatment. Operative treatment is indicated and should be resorted to without undue delay. Simple plastic procedures on the constricted area of the bowel are as difficult as, and usually less effective than, a short-circuiting entero-enterostomy or resection. We believe that entero-enterostomy is the treatment of choice in this type of benign localized obstruction.

Decision to perform an entero-enterostomy at the time of the initial operation for relief of a strangulated hernia to obviate a future stenosis is a matter of surgical judgment which must be decided upon the merits of each individual patient. Watson stated that the constricted area should relax when this portion of the bowel is

stroked gently with a moist pledget of cotton. If the groove persists, it signifies a reasonable prospect of future stenosis and should be treated accordingly. It is, of course, the essence of wisdom to remember at all times that the primary purpose of emergency surgical intervention is to save the patient's life and that considerations of definitive cure are not to take precedence over this primary concern.

Serious complications of intestinal intubation, directly referable to the tube, are rare. Smith, in an analysis of 1,000 cases involving the use of the Miller-Abbott tube, listed eleven instances of serious sequelae, including hemorrhage, ulceration and perforation of the bowel. Iglaeur and Molt reported several instances of laryngeal edema requiring tracheotomy. Knotting of the tube, overdistention of the balloon and many minor complications have been observed in the use of the Miller-Abbott tube. Intussusception of a sufficient degree to cause obstruction of the bowel, such as occurred in one case reported herein, is a rare complication of intestinal intubation. A similar example has not been found in the literature. Presence of this complication was not appreciated in this instance, and a fatality resulted.

CONCLUSIONS

Stricture of the small intestine may occur following reduction of a strangulated hernia.

Future stenosis may be anticipated and prevented at the time of the original operation in some instances.

Intussusception of the small intestine over an indwelling Miller-Abbott tube may occur and result in complete intestinal obstruction.

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MULTIPLE DIVERTICULA OF THE JEJUNUM

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DIVERTICULA of the jejunum constitute the rarest type of diverticulum which occurs in the gastrointestinal tract. It is exceedingly rare in comparison with diverticulosis of the duodenum and the colon. This condition was first described by Sir Astley Cooper in 1844 when he found numerous pouches of the jejunum in a postmortem examination upon a man of sixty-five. In 1881 Dr. Osler, in a postmortem examination upon a man of fifty-five, reported diverticula varying in size from a cherry to a large apple. Many other men have described this condition. Hansemann found 400 diverticula in a man of eighty-five at a postmortem examination. Dr. Harold C. Edwards, King's College Hospital, London, in a book published in 1939 gives the most extensive and interesting descriptions of various diverticula of the small and large bowel, and he states that he studied sixteen cases of diverticula of the jejunum, eight of which were from postmortem examinations, four from operations and four from x-ray records. Rankin and Martin in 1934 and Chapman in 1933 and 1934 contributed excellent papers to this subject. Sheppard collected sixty-one cases up to 1935. Levy and De-Groat collected sixty-six cases up to 1935. Rosedale and Lawrence collected seventy-eight cases up to 1936 and reported three cases in 5,000 postmortem examinations. Goddard reported sixteen diverticula of the small intestine (other than Meckel's diverticula) in 13,069 autopsies. Up to 1938 Gerstler found 187 cases in the literature. Since then Ravenswaay increased the number of cases to 228. From a roentgenologic standpoint no accurate figure is possible because they are so easily missed. Rosedale and Lawrence did not find a single example in 5,000 complete gastrointestinal examinations. Rankin and Martin state that jejunal diverticulosis is found once in 25,000 x-ray examinations. Spriggs and Marxer found thirteen cases in 1,000 radiographs. Johns in 1937 said only twenty-six cases had been discovered by x-ray and only seventeen of these were confirmed by operation. Weber states that if the roentgenologist is given the opportunity, he can diagnose the condition by roentgenoscopic and roentgenographic examination if characteristic roentgenograms are produced.

Benson, Dixon and Waugh in 1943 made the statement that such diverticula are not exceedingly rare. They stated that the Mayo Clinic, from 1909 to 1942, observed diverticula of the small intestine in 122 cases. These 122 cases included the fifty-two cases that were reported in 1934 by Rankin and Martin. In addition to the 122 cases reported by the above observers, approximately 200 other cases are recorded in the literature.

The records of the Maryland General Hospital, Bon Secours Hospital and South Baltimore General Hospital were searched for the history of a case with multiple diverticula of the jejunum, both in the hospital and x-ray departments, but none was found. The records of the University of Maryland Hospital were reviewed and it was found that one patient, aged seventy-six years, was admitted and found to have two dozen diverticula of the jejunum. Another patient, aged forty-five, who was admitted on the medical service, was found to have several small diverticula of the jejunum. No operative procedure was performed. The patient's symptoms were epigastric distress, fullness following meals and a smothering sensation. The patient complained of gaseous distention for the past two years, some nausea and the desire to vomit.
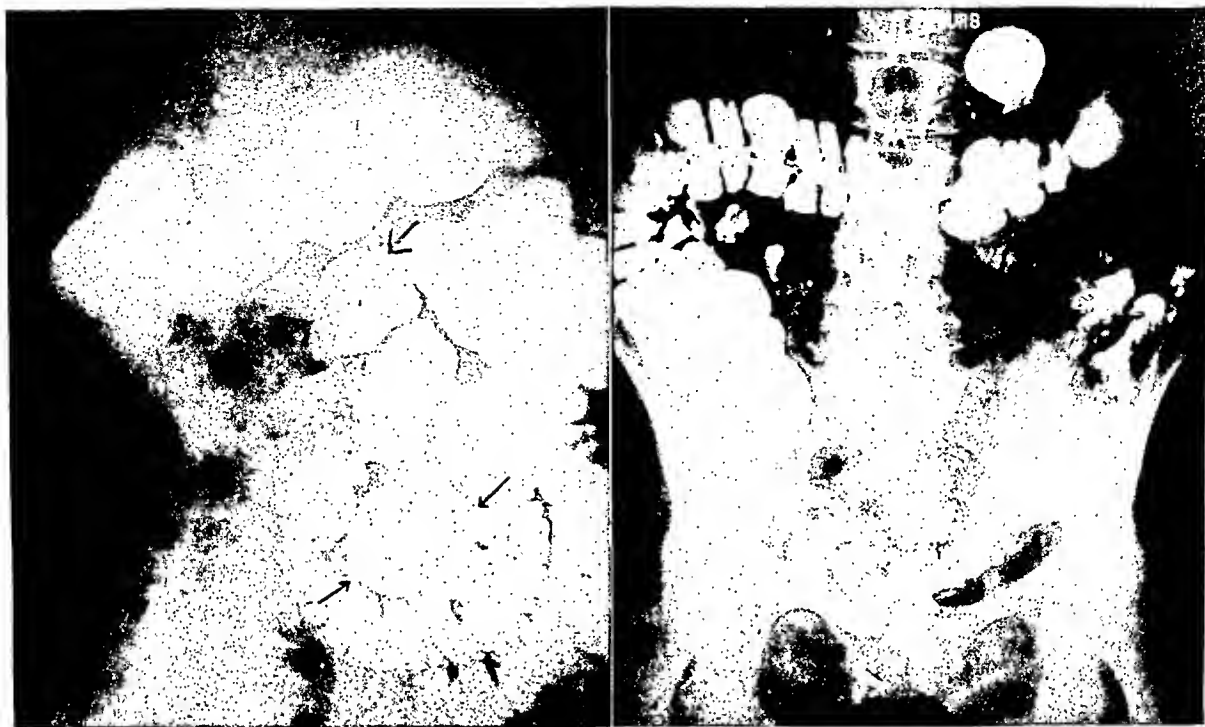
FIG. 1. A white male, aged eighty-one, a case of Dr. Charles Waters, showing large diverticula of the jejunum. No operation was performed. The patient died several years later from other causes.

FIG. 2. A white female, aged seventy-eight, a patient of Dr. Charles Waters, with multiple diverticula of the jejunum; no operation was performed and the patient is still living.

Dr. Charles Waters, a radiologist in Baltimore, had two patients in his experience who had multiple diverticula of the jejunum but no operation was performed. (Figs. 1 and 2.)

Most diverticula of the small intestine are of the mucous membrane, hernia type. Practically all are of the acquired type. The site of herniation of the mucous membrane through the wall of the intestine occurs at the area where the blood vessels perforate the muscular coat of the intestine. Most observers believe that two factors are present: (1) a weak area in the bowel wall and (2) a pulsion force acting from within the bowel which starts the process of herniation. The protrusion occurs where the blood vessels perforate the muscular coat of the intestine. There are some observers, however, who believe that they may be congenital in origin. Of the 122 cases in the Mayo series, 100 cases occurred in the jejunum. In seventeen of their cases the diverticula were limited to the ileum,

and in five cases the diverticula occurred throughout the small intestine. While these diverticula may occur in any part of the circumference of the bowel, a majority of them are situated either within the leaves of the mesentery or along the mesenteric attachment. The diverticula of the jejunum are much larger than the usual tiny diverticula seen in the colon. The mouths of these diverticula may vary from 2 or 3 mm. to 3 or 4 cm. in diameter. In forty-four of the 122 cases reported by Benson, Dixon and Waugh only one diverticulum was observed. In sixty-six cases three or more were present. Diverticula in other viscera were observed in forty-nine of eighty-five cases in which autopsy was performed. In these reports they state that these diverticula are noted in all age groups, seven cases being found under ten years of age. The youngest in their present series of cases was twelve years old. The oldest was ninety-one. At the Mayo Clinic most of the diverticula of the small intestine

have been found at autopsy—(eighty-five cases). Twenty-one cases were recognized during the course of abdominal operations. They state that these diverticula are not easily identified at the operating table because they offer so little resistance to the palpating hand that they may escape identification, and often they are hidden within the leaves of the mesentery. In sixteen of the 122 cases the diverticula were found on x-ray examination of the small intestine.

The symptoms of chronic intestinal obstruction may be associated with diverticulosis of the small intestine. This was the most commonly observed complication in most of the cases reported. The more marked symptoms are (1) vague pain or abdominal discomfort and (2) flatulence. The flatulence is due to retention of intestinal contents in the diverticula. The pain may be due to the same cause or, if pain is particularly severe and colicky in nature, it may be due to obstruction in the bowel by a large pouch. Other complications such as acute obstruction, inflammatory disturbances resulting in gangrene and perforation, hemorrhage from the diverticula, rupture of the diverticula, foreign bodies and neoplastic diseases may be encountered.

Treatment. If the patient is asymptomatic and there are no complications, surgery is not required. These people may be carried along satisfactorily by proper control of their bowels and limitation of intake of foods rich in starch and cellulose. When the diverticula are of large size and x-rays show a long retention of barium and when the patient has symptoms of chronic intestinal obstruction, the treatment should be surgical and the proper surgical procedure should be resection. The resection can be done either by end-to-end anastomosis or by lateral anastomosis.

CASE REPORT

An interesting case of multiple diverticula of the jejunum came under our care:

A white female, aged sixty-three, entered the Maryland General Hospital on January 5, 1943. She complained of generalized abdominal pain and marked distention. The past history showed that the patient had had her gallbladder and appendix removed in this same hospital by another surgeon fourteen years before. At that time she had pain in the upper abdomen with jaundice and the gallbladder contained many stones. No mention was made in the operative note at that time of any pathological condition involving the jejunum. In 1938, four years prior to admission, the patient had had a Mayo hysterectomy performed in another hospital where a diagnosis of prolapse of the uterus, cystocele and rectocele had been made. The patient dated her present illness to three years prior to admission when she began to have cramp-like pains over the entire abdominal area. These attacks of pain bore no relation to the ingestion of food although the patient said she felt better when she remained on a vegetable diet. She stated that she had twelve to fifteen semi-solid stools per day but noticed no blood or mucus. She had had no jaundice since 1929. In the last few months the abdominal pain had been more severe in character and there had been an accumulation of gas in the intestines which was difficult to relieve. Three years before she weighed 190 pounds and since then had lost 62 pounds. In the last few weeks she had had difficulty in moving her bowels. She had had some belching and nausea associated with attacks of vomiting.

Physical examination on admission revealed a sixty-three year old, white female, showing evidence of recent loss of weight, at this time weighing 128 pounds. The abdomen showed a well healed upper right rectus scar with no hernia present. The muscle tone of the abdominal wall was poor. Many loops of distended bowel could be seen and easily palpated through the abdominal wall. No masses were palpable. The liver was not enlarged. There was no fluid in the abdomen. Pelvic examination showed a good perineal floor. The uterus and adnexa were absent or at least could not be palpated. The blood pressure was 180/90. No evidence of cardiac disease was present. Rectal examination, both digitally and by proctoscopic examination, revealed no lesion in the lower bowel.

X-ray examinations of the chest, both fluoroscopic and with films, showed the lung

FIG. 3. Mrs. E. H., radiograph showing position of diverticula in jejunum below the stomach and transverse colon.

fields to be clear with normal root and bronchial markings. The heart measured 12¼ cm. across its greatest transverse diameter with an internal diameter of the chest of 26 cm. A barium enema showed that the bowel filled without difficulty but was somewhat redundant. (Fig. 3.) Two or three fair-sized diverticula were found in the sigmoid area. There was no filling defect to suggest malignant growth.

The stomach was reported to be slightly taut and pulled to the right side. It was atonic but no defect was found in the stomach, the prepyloric area or the duodenum. No evidence of new growth was found nor was there evidence of any pathological condition in the small or large bowel.

The blood chemistry, urinalysis, stool examination and blood studies showed no abnormality. The Wassermann test was negative. A tentative diagnosis of intestinal obstruction involving the small bowel was made. From the symptoms and signs it was believed that the obstruction was of a chronic nature and had periods in which the patient was much better and at other times much worse. Because of great loss of weight, and the more marked symptoms that the patient had in recent weeks, it was believed that abdominal exploration was indicated.

Under spinal anesthesia an upper midline incision was made. All of the abdominal organs were found to be normal except the jejunum and the descending colon. There were a few diverticula in the large bowel. The jejunum was found to be markedly distended and hypertrophied, being twice the normal diameter. Beginning about 6 inches below the ligament of Treitz, there were multiple diverticula along the mesenteric side of the bowel, varying in size from a marble to a small orange. These diverticula extended for a distance of 3½ feet.
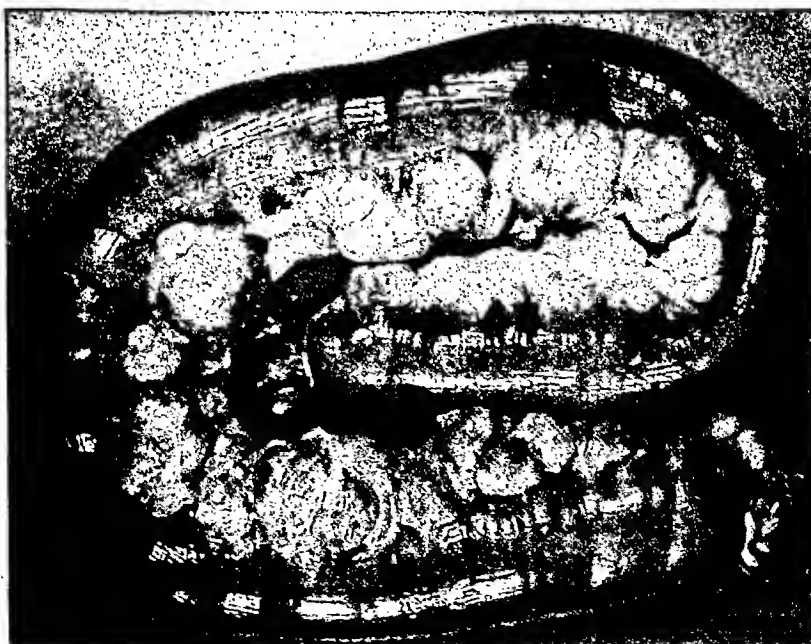
FIG. 4. Specimen removed at operation showing multiple diverticula.

There was no definite demonstrable mechanical obstruction of any portion of the jejunum. Some of the diverticula were very large and seemed to compress the bowel, but when squeezed, the gas passed readily into the intestine. In view of the patient's history of marked weight loss and her other symptoms and since this was the only pathological condition found, it was deemed advisable to resect this entire portion of jejunum. (Fig. 4.) Therefore, about 3½ feet of jejunum were removed between Payr clamps. The ends of the bowel were closed and inverted. The proximal portion of jejunum was anastomosed to the distal portion of jejunum by lateral anastomosis with rubber-covered intestinal clamps. The abdomen was closed and the patient returned to bed in excellent condition.

The specimen removed consisted of a section of jejunum well over a meter in length which, by actual count, contained fifty-nine diverticula. These diverticula varied in size from a medium-sized marble to a small orange and were situated at or near the mesenteric border of the intestine.

Gross Diagnosis: Multiple diverticula of the jejunum.

Microscopic examination showed the walls of the bowel to be somewhat hypertrophied, but the diverticula contained thinned-out muscle fibers with nothing but serosa and a thin layer of mucosa. (Fig. 5.)

After operation, when reporting to the roentgenologist of what was found, we reviewed the x-ray plates and then saw diverticula of the jejunum which we had missed originally.

The patient made an uneventful recovery and was discharged three weeks after operation. While in the hospital, she gained 5 pounds in weight. The patient has been seen several times during a period of three and one-half years. One month before the preparation of this report she was in excellent health and weighed 170 pounds. She had had no digestive disturbances and no stool abnormalities since operation three years and ten months before.

SUMMARY

As far as I am able to determine by existing records, 322 cases of jejunal diverticula have been reported previously in the literature. A great majority of these cases were reported as result of autopsy findings or

Fig. 5. Microscopic appearance of the wall of one of the diverticula showing thinned out muscle fibers with a thin layer of mucosa and serosa.

x-ray examinations of patients with the condition. A new case, that of a sixty-three year old female who was successfully operated upon, is added to this list. The condition can and should be diagnosed by x-ray findings. There are many cases which give rise to no symptoms, but those patients who produce symptoms of obstruction or other complications should be operated upon for relief. The best type of operation is resection.

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IN some cases of pyloric obstruction *all* tests, including x-rays, gastroscopy, etc., fail to establish firmly the correct diagnosis, according to Segal et al. The authors state that they have used sodium amytal often with success in trying to differentiate pyloric obstruction due to intrinsic causes (ulcer, fibrosis, carcinoma, etc.) from persistent pylorospasm of psychogenic origin. (*Richard A. Leonardo, M.D.*)

PREGNANCY COMPLICATED BY TRANSMESENTERIC HERNIA

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THE association of pregnancy and acute intestinal obstruction from any cause is fortunately a rare occurrence. A number of references to this combination can be found in the German literature in contrast to the few references to the topic in the English.

A series of cases have been collected by Ludwig,[3] von-Mikulicz-Radecki[7] and Eliason and Erb.[2] The prime causes of obstruction are seen to be adhesions, volvulus and the pregnancy itself where the simple enlargement of the uterus produces mechanical obstruction of the bowel by external pressure. (Table 1.)

TABLE I

STATISTICAL SUMMATION OF THREE SERIES OF PREGNANCIES COMPLICATED BY INTESTINAL OBSTRUCTION

	Ludwig	von Mikulicz-Radecki	Eliason and Erb	Total
Adhesions	28	15	14	57
Volvulus	13	34	11	58
Pregnancy Certain	10	10	28	48
Uncertain	0	8	0	8
Kink in the arteries of the mesentery	1	0	0	1
Tumors and cysts	25	4	7	36
Obturation	7	0	0	7
Intussusception	4	3	0	7
Hernia	7	1	2	10
Paralytic inflammatory	0	0	4	4
Unknown	0	6	0	6
	95	81	66	242

A study of the maternal mortality in these three series of cases is interesting as it reflects the progress of surgery during these periods. In the patients of Ludwig, end results were known in eight-nine cases. Forty-nine patients died, giving a total mortality of 55 per cent. Von Mikulicz-Radecki was able to determine the end results in seventy-one of his collected cases. Twenty-eight patients died, resulting in a mortality of 39.4 per cent. In their sixty-six patients, Eliason and Erb report fourteen deaths and a mortality of 21 per cent.

The patient, whose case history will be detailed later, had a transmesenteric or internal hernia. Intra-abdominal hernias producing acute intestinal obstruction are unusual entities. Only seven internal hernia were found in an analysis of 136 cases of acute intestinal obstruction encountered at the Mayo Clinic from 1935 to 1939, inclusive, by Mayo, Stalker and the author.[4] A review of all types of internal or intra-abdominal hernias at the same clinic reported by the same authors[5] revealed that only thirty-nine such cases were encountered in the years 1910 to 1939 inclusive.

These hernias protrude into an abdominal pouch or opening of the peritoneum or through an aperture in one of the several mesenteries, in contrast to external hernias in which abdominal contents pass through defects in the wall of the abdomen. An internal hernia may be primary, a direct result of some congenital defect or secondary to operation, trauma or or inflammation. Thus, they include: (1) Those which occur in normal pockets such as the foramen of Winslow and the paraduodenal, paracecal and intersigmoid fossa; (2) those occurring in exaggerations of normal mesenteric folds as those in the broad ligament of the uterus; (3) those resulting from traumatic or operative bands or adhesions; (4) those resulting from chronic inflammation and (5) those which occur in anatomic defects.

Internal hernias due to defects in the normal mesenteries, inadvertently created by the surgeon, are infrequently seen now owing to the proper observance of technic at the time of performance of an operative procedure. Therefore, the more important group at present are the internal hernias

resulting from congenital defects. Although few in number, such hernias assume considerable clinical significance in consequence of the severe degree of acute intestinal obstruction with which they are associated.

Transmesenteric hernias are placed in the group owing to anatomic defects. The hernia which is reported in this paper belongs in a subvariety of this group and was due to a prolapse of the ileum through an aperture in its mesentery. The cause of the aperture in these cases may be either congenital or the result of a thinning of the mesentery with eventual tearing on an inflammatory or a traumatic basis. The great majority of defects in the mesentery of the small intestine are located close to the ileocecal valve although they may be found proximally in the mesentery of the ileum or jejunum. A hernial sac is not present as the intestine merely prolapses through an aperture in its own mesentery.

These patients are acutely ill; the serious nature of their illness may be more fully appreciated from the total mortality of 38 per cent which Cutler and Scott[1] report in their review of fifty patients having transmesenteric hernias but which were not associated with pregnancy. The recognition that acute, intestinal obstruction is present is far easier than the making of an exact etiologic diagnosis. Cutler and Scott state that the specific diagnosis was never made correctly before laparotomy in any of their reported cases. Consideration of the problem emphasizes that there is little to differentiate the presence of an incarcerated, transmesenteric hernia from other types of serious intraabdominal pathologic conditions.

The superimposition of an acute internal herniation upon an existing pregnancy produces an interesting, complex diagnostic problem which is difficult to unravel in the preoperative period. The recognition that an acute abdominal catastrophe has occurred must, of course, lead to laparotomy and the mechanical correction of the difficulty. The pregnancy should not be a deterrent factor in treatment and the reaction of a state of watchful expectancy.

A situation similar in many respects to the one which is to be reported in this paper was reported by Rochard[6] and treated by Morestin. The patient was a twenty-nine year old, white female who, four years prior to the episode reported, had an attack of abdominal pain which was attributed to appendicitis. At this time, surgery was not performed. One year later, the patient became pregnant and was delivered normally. She subsequently became pregnant again and, as she drew near to term, she exhibited many of the symptoms and signs of eclampsia; however, undergoing a spontaneous labor, she was delivered normally of a living infant. Shortly after delivery, the patient complained of abdominal pain; nausea and vomiting occurred and became persistent. Morestin saw the patient about twenty-four hours after delivery. At that time, her abdomen was distended and a tumor was palpated to the left of the umbilicus in the midline. The tumor was round, mobile, tender and tympanitic to percussion. Morestin thought that an intestinal obstruction due to a volvulus was present and advised immediate laparotomy. At operation, he found that a loop of small intestine had passed through a hole in its own mesentery and then had undergone volvulus. The volvulus was easy to untwist and after enlarging the aperture, through which the small intestine had prolapsed, Morestin was able to effect complete reduction. Resection of the involved portion of the bowel was not necessary. The patient made a good recovery.

CASE REPORT

The patient was a married, white female, aged thirty who was seen early in pregnancy. She stated that she had had severe pain of sudden onset in the right lower quadrant of the abdomen for about five hours. Nausea and vomiting of a marked degree were present. The patient stated that she had never had any such attacks previously. Abnormalities of urination had not been present. The menstrual history was interesting in that the patient missed her

last period which should have occurred six weeks prior to the onset of her illness. Vaginal bleeding had not been noticed since her last period and she had never been pregnant before. A detailed review of the other systems did not contribute anything of importance to the history.

Her temperature was 98.2°F., the pulse rate, 72 per minute and the respiratory rate, 14 per minute. Physical findings of a positive nature were limited to the abdomen. Peristalsis was not seen and a mass was neither seen nor palpated; a moderate degree of muscle spasm was present over the entire abdomen. Direct and rebound tenderness were present in all quadrants of the abdomen being most marked in the right lower quadrant. External hernias were not present. Bimanual vaginal examination revealed the cervix to be normal; a marked degree of tenderness was present in the right lateral fornix and a moderate degree in the left. Similar findings were found upon rectal examination.

The erthroeyte count was 3,870,000 per cu. mm, the value for hemoglobin 76 per cent and the leukocyte count 21,900 per cu. mm. of which 90 per cent were found to be polymorphonuclear neutrophilic leukocytes. Urinalysis revealed a trace of sugar but the remainder of the examination was within normal limits.

It was quite evident that the patient had suffered some acute abdominal accident of an unusual nature. The insult to the peritoneum had been great and preeipitous as evidenced by the short duration of the illness, the physical findings and the leukocyte and differential counts. The situation presented was not that of the usual case of acute appendicitis. Considered among the preoperative diagnosis was some type of internal hernia, volvulus and, in view of her menstrual history, a right, ruptured, tubal, ectopic pregnancy.

In any event, immediate laparotomy and abdominal exploration were in order. The abdomen was opened through an incision in the right rectus abdominis muscle. Upon opening the peritoneum many coils of gangrenous ileum, which were twisted upon themselves, were found. At first view, it was thought that a simple volvulus of the terminal ileum was present but an attempt at untwisting the bowel was fruitless. The base of the knot was completely visualized and the bowel was seen to have prolapsed through an aperture in the mesentery of the ileum from the upper to the lower side. The aperture was about 2 cm. in diameter and about 30 cm. from the ileocecal valve.

The involved area of small intestine was irrevocably gangrenous. Reduction of the hernia was not done since it would have been extremely difficult and without ensuing advantage if accomplished. The conservative procedure and the one of choice was exteriorization and resection of the involved area; therefore, the cecum and a small portion of the ascending colon were mobilized. The right ureter and ovarian blood vessels were visualized and not injured. The mesentery of the involved area of the ileum was severed below the aperture of herniation until an area supplying viable small intestine was reached. This procedure neeessitated the resection of about 130 cm. of ileum. The denuded area was recovered with peritoneum and the small and large intestines prepared for subsequent union by suture of the two loops in a side-to-side position. The ileum, cecum, appendix and that portion of the ascending colon which were to be removed were then brought out of the abdomen and a three bladed Rankin clamp applied. At this time, gentle palpation of the uterus revealed it to be soft and slightly enlarged. The adnexal tissues on both sides were normal.

Two Penrose drains were placed and brought out through a stab wound in the right flank. The exteriorized portions of the small and large intestines were placed at the upper end of the incision and the peritoneum and the anterior layer of fascia of the rectus abdominis muscle were sutured. The skin at the upper and lower angles of the wound was then approximated by interrupted dermal sutures. Large curved hemostats were placed on each end of the exteriorized area, just above the Rankin clamp, and the affected portions of the small and large intestine resected. Powdered sulfathiazole was then placed around the area of amputation.

The course and treatment of the patient, subsequent to operation, were not unusual. Parenteral fluids and vitamins were administered, gastric suction was employed and a transfusion of citrated whole blood was given. Adequate amounts of sulfathiazole were also given. On the second day after operation, an opening was made in the ileum just below the Rankin clamp and a catheter was inserted and secured by two purse-string sutures. This pre-

vented contamination of the wound for several days. Subsequently, the catheter came out and the Rankin clamp came away spontaneously. The spur between the proximal and distal loops was crushed in preparation for closure of the ileocolonic stoma. This was accomplished successfully at a later date. A Friedman test for pregnancy was done during the early part of the hospital stay of the patient and was reported as positive. This confirmed the preoperative suspicion of pregnancy and accounted for the soft, enlarged uterus found at operation. The patient had a normal ante partum course later being delivered of a living male.

Four years have passed since this patient was subjected to surgery; in the interim she has remained well. A mild diarrhea was noted during her early convalescence and this has persisted irregularly to the present. Recently, a barium enema was administered . and a roentgen study of the colon was done. This revealed a normal pattern of the mucosa of the colon and the present terminal ileum with the operative defect described previously.

The operative procedure of choice when the involved area is in the lower ileum and cecum is simple exteriorization and resection. Restoration of intestinal continuity should be deferred until a later date. Exteriorization can be performed fairly rapidly with a minimal of trauma to a patient who is already seriously ill and unable to withstand prolonged surgery. Cutler and Scott report a mortality of 20 per cent in five patients with resection of the Mikulicz type. This figure stands in favorable contrast to a mortality of 35.2 per cent in seventeen patients in whom resection and primary anastomosis was done. If the proximal portion of the ileum or jejunum is effected, it may be necessary to do a resection with primary anastomosis.

Treatment of the pregnancy itself should be entirely conservative. Abortion or miscarriage may ensue but the treatment of the pregnancy should be conservative and entirely along general surgical principles unless complications which threaten the life of the mother develop. Then evacuation of the uterus must be given serious consideration.

SUMMARY

Acute intestinal obstruction appearing during pregnancy is uncommon. An internal hernia producing acute intestinal obstruction is rare. A patient who was early in pregnancy and who suffered an internal hernia was successfully treated surgically and was later delivered of a normal, living male.

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PRIMARY MALIGNANT MELANOMA OF FEMALE URETHRA

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MALIGNANT melanoma of the female urethra has been reported at very infrequent intervals and the total number of cases in medical literature is small enough to make an added case of some interest. Its infrequency is well illustrated by the. fact that the Mayo Clinic had only three cases to report from 1907 to 1945. During this period 700,000 female patients were examined and seventy-seven primary carcinomas of the female urethra were found, of which only three were primary melano-epitheliomas.

The nomenclature of this pathologic entity is somewhat confusing since there is still some difference of opinion as to the theory of origin of malignant melanomas. There are those who consider it a sarcoma, and there is also the school that considers it of epithelial origin, hence a melano-epithelioma. We believe that malignant melanoma is of epithelial origin and is usually derived from a pre-existing nevus.

In 1896, Reed reported the first authentic case of primary melano-epithelioma of the female urethra. Since then or approximately within a period of fifty years, thirteen other cases were reported.

The disease is manifestly one of old age, the average age of the collected cases being sixty-four years. The youngest patient, reported by Kyrle in 1940, was thirty-two years of age and the oldest was seventy-eight, reported by Long, Counseller and Dockerty. In Kustner's case the age was not given. The age of the patient in this case report is seventy and has been included in the computation of the average. The patients usually complain of vaginal discharge, bleeding and urinary symptoms such as frequency and burning. Urinary obstruction is an unusual symptom.

Grossly the tumor appears pigmented and varies in shade from black to blue with a tendency to spread by superficial lymphatics or through the blood stream. Metastases are widespread and this consequently greatly enhances its deadliness. They frequently are first noted under the skin where they arrange themselves as small, fine nodules which may or may not be pigmented. Malignant melanomas of the eye spread by the blood stream and not through the lymphatics.

Histologically, the cells may be elongated and spindle-like so as to give the impression of fibrosarcoma. In other instances and most frequently, the cells are arranged in an alveolar grouping which strongly suggests an epithelial tumor. The cells are usually large, clear and polyhedral with a large vesicular nucleus and a pale cytoplasm. One section of a slide may impart an impression of carcinoma, another of sarcoma and still another of endothelioma. Mitotic figures are usually in evidence.

The treatment of malignant melanoma is uniformly and consistently unsatisfactory. Surgical excision usually suffices to eradicate the local lesion. However, it is not long before it recurs elsewhere by fatal metastasis. X-ray and radium therapy have been equally disappointing. Handley recommended radical excision with the neighboring glands and intervening lymphatics. In malignant melanoma of the female urethra this obviously cannot always be done.

Newell and Schrivner report a case in which the growth was removed ten times. It was cauterized with actual cautery five times, implantation of 50 mg. of radium directly into the urethra for eight hours on four different occasions and deep x-ray therapy (200 KV) on one occasion. In each instance the growth recurred within two or three months.
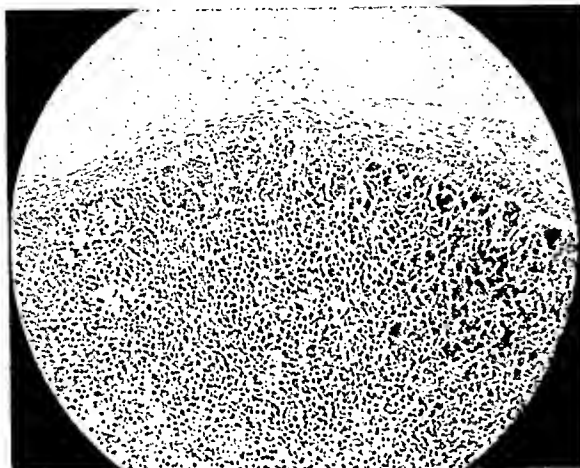
FIG. 1. Low power magnification of tissue removed from urethra at operation.
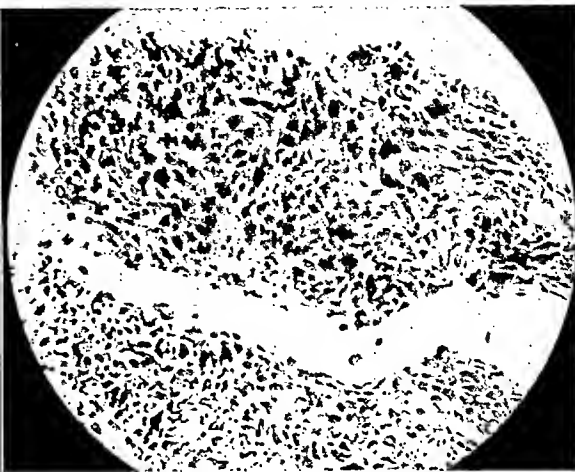
FIG. 2. High power magnification of tissue removed from urethra at operation.

CASE REPORT

On March 5, 1942, a seventy year old female patient (J. D.) was admitted to the hospital with the chief complaint of bleeding from the vagina. Her family and past history was irrelevant except for the history of three operations for "vaginal tumor" in 1933, 1929, and 1925. These operations were done in a physician's office and no record could be obtained.

About two months prior to admission, she began to complain of vaginal bleeding and burning and general discomfort which was aggravated and exaggerated during micturition. She had frequency and nocturia. The bleeding and pain became more pronounced with the passage of time.

Physical examination was essentially negative except for a bright red and hemorrhagic appearing mass which was found protruding from the urethral orifice. The mass was cauliflower in appearance and bled easily on pressure. It was approximately the size of a filbert nut.

At operation the mass was excised from the posterior urethra and the bed sutured. At this time it was noted that the mucosa of the urethra was bluish in appearance. Bleeding was easily controlled and an inlying catheter was inserted.

The immediate postoperative course was quite uneventful and she was discharged from the hospital in five days much improved. She was seen on several occasions and seemed to be quite well.

The report of the pathologist (C. E. Schradieck) follows: "The specimen consists of two tabs of tissue of soft consistency and of dusky-livid color designated as caruncula urethralis. Each piece covers an area of a square centimeter and is of about $\frac{1}{2}$ to $\frac{3}{4}$ cm. in thickness. Both pieces are somewhat lacerated. The surface appears somewhat granular and papillary.

"In part, the growth is covered with cutaneous epidermis. The papillae of the underlying stroma are distended with densely crowded round and polyhedral cells. The stroma between these cell groups is scant and, owing to edematous and in part hemorrhagic infiltration, is not clearly outlined. The cells infiltrating it show scant cytoplasm and rounded dark staining nuclei. A few show granules of brownish pigment (melanin). Some such cells are also seen within the basal layer of the epidermis. Mitotic figures are practically absent. In some areas these cells are densely crowded but do not appear to form a strictly coherent tissue. Prickle cells were not seen. In other areas the cells are more loosely strewn. Extravasated blood is seen between the cell groups here and there. In some areas a few of the cells contain melanotic pigment but most of them do not. A few pigmented chromatophore cells of mesoblastic character were also scattered here and there. The diffusely infiltrating manner of growth, the irregular sprinkling of it with chromatophores and melanoblasts, the polymorphic character of the cells which are in some areas short spindle and in others more aplastic agglomerations of rounded and polyhedral cells are suggestive of malignant melanoma. These cells are quite densely apposed on the whole and loosely strewn through the stroma in other areas with peritheliomatous grouping about thin-walled blood

vessels or about lymphatic spaces. The above, together with the diffuse penetration of the subepidermoid stroma, convey the impression of malignant melanoma, originating apparently from nevus tissue within the excised area." (Figs. 1 and 2.)

About one year later, subcutaneous nodules became palpable and visible in the inguinal regions and on the abdomen. There was no evidence of local recurrence. These nodules were dark blue and brown in appearance, and were slightly tender upon palpation. Ophthalmoscopic examination of the fundi showed the usual changes associated with senescence. After July, 1942, direct contact with the patient was lost due to the fact that one of us (J. S.) entered the Armed Forces. In September, 1942, she was hospitalized at another institution where she was observed for several days and discharged as having generalized carcinomatosis. No biopsies or x-ray examinations were performed at that institution. She expired on October 2, 1942, nineteen months after operation. No autopsy was performed.

The authors are indebted to Dr. B. Earl Clarke for his kindness in preparing the photographs of the microscopic sections.

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LIGATION OF THE INFERIOR VENA CAVA AND OVARIAN VEINS FOR INFECTED ABORTION*

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THE treatment of infected abortion is conditioned by the pathologic picture presented by the patient upon admission to the hospital. Falk's classification[1] of types is very useful: (1) infection limited to the uterine cavity; (2) parametritis; (3) thromboplebitis; (4) abscess of the wall of the uterus; (5) general sepsis; (6) generalized peritonitis; and (7) perforation of the uterus. This presentation is concerned purely with the treatment of a patient with infected abortion with parametritis, thrombophlebitis and pulmonary infarction, a mixed type two and three.

Early diagnosis of pelvic thrombophlebitis is not always easy. Pulmonary emboli furnish definitive evidence of their presence. Recurring, spiking temperatures with chills are also diagnostic but phlebitis with embolization may occur without this characteristic temperature curve. Manual palpation of thrombosed pelvic veins is only an occasional finding when the phlebitis is secondary to an infected abortion commonly masked by a surrounding parametritis. Not infrequently, patients are observed who present evidence of pulmonary emboli secondary to thrombosed pelvic veins due to abortion without any abnormal findings on pelvic examination. The author would also like to emphasize the possibility of a low grade pelvic peritonitis with few or no clinical signs; conversely, hypogastric tenderness and mild spasticity may be produced by parametritis without gross peritonitis.

Therapy for pelvic thrombophelbitis of uterine origin has run the pendulum of surgical and non-surgical approaches. The latter relies primarily upon chemotherapy and transfusions to sterilize the lesion.

Cures of proved pelvic thrombophlebitis with embolization have resulted under this regimen. The more radical school operates on the well established theory that ligation or excision of involved veins prevents venous dissemination of infection. The operative treatment of the patient under discussion must be directed at the ovarian veins as well as those draining the uterus. Nelson et al.[2] found the ovarian veins affected in eighteen of forty-one cases of proven pelvic thrombophlebitis. Hysterectomy, leaving behind many thrombosed parametrial veins, is insufficient as a solitary procedure. The internal iliac veins, common iliac veins or the inferior vena cava below the renal veins must be ligated. The author believes caval ligation is technically the easiest and safest of the three.

The transperitoneal approach permits ligation of both ovarian veins and the inferior vena cava through a single incision. The extraperitoneal approach requires bilateral incisions but is much less shocking and is unaccompanied by the postoperative complications attending a laparotomy. The transperitoneal approach was employed in this case because, along with the thrombophlebitis, a low grade peritonitis was suspected for which hysterectomy as well as vein ligation is necessary. This patient developed postoperative complications, avoidable through the extraperitoneal approach. She was a Negro, a race which has a greater tendency to develop adhesive peritoneal reactions to laparotomy. In future cases of this type, it would be more desirable to follow Krotoski's suggestion[3] of staged procedures. He recommended extraperitoneal ligation of the

inferior vena cava and the right ovarian veins at the first stage and, if symptoms persist, a second stage wherein the left ovarian vein is ligated extraperitoneally. In complicated cases, hysterectomy may be necessary as a third stage.

Collins et al.[2,4] have reported eight consecutive transperitoneal ligations of the inferior vena cava and both ovarian veins with only a single death. Their reports included protocols of three successful cases and of these, only one was a case of thrombophlebitis secondary to infected abortion. This case substantiates their finding that a seemingly radical operation may be performed for this condition with a cure. ·The author believes, however, that at present, insufficient statistical figures are available to compare surgical with non-surgical therapy for this specific pathologic type of infected abortion.

CASE REPORT

M. S., a twenty-five year old negro nulligravida, was admitted to the Gouverneur Hospital on August 11, 1946. Her past history was negative except for an appendectomy in 1943, followed by intestinal obstruction which required re-operation and lysis of adhesions two weeks postoperatively.

Her menses had always been regular and she had a normal period between April 29th and May 6th. She did not menstruate in June and on July 19th induced abortion by instilling undiluted lysol into her uterine cavity. This was rapidly followed by vigorous bleeding and passage of large clots and tissue. The bleeding persisted for two days to be succeeded by a brownish, foul vaginal discharge. Between July 21st and August 4th the patient suffered mild, lower abdominal pains accompanied by febrile symptoms. Throughout the week of August 4th to August 11th, these pains were overshadowed by severe pain in the right hip which made walking intolerable and it was with this complaint that she entered the hospital.

On admission, her temperature was 104°F., pulse 120 and respirations 18. It was rapidly determined that her symptoms were not produced by intrinsic disease of the hip for passive motion of the thigh in all directions was pain-

less and x-ray of the hip revealed no abnormalities. Vaginal examination disclosed a tender, boggy, slightly enlarged anteverted uterus; the cervix was patulous to the tip of a finger. Both parametrial regions were densely indurated and exquisitely tender. The cul-de-sac was free and the adnexa were not palpable. There was a foul, brownish vaginal discharge. Physical examination of other systems revealed no abnormalities. Laboratory tests indicated that her urine was normal; hemoglobin, 9.5 Gm., white count, 14,000 and Wassermann, negative. The diagnosis of postabortal parametritis was made and the patient was placed on a course of intramuscular penicillin.

On the day after admission, she complained of sudden pain in the right chest anteriorly and expectorated bloody sputum. For the week following admission she ran a stormy course, complaining of recurrent pains in both chests of deep inspiration and expectorating almost frank blood. Her temperature fluctuated between 102 and 104°F., her pulse between 120 and 140, with respirations averaging 24. On August 17th, the patient had a chill and a rise in temperature to 105°F. An x-ray of the chest taken on August 13th, revealed a soft opacity in the right upper lobe field and one taken two days later disclosed multiple areas of opacity over both lung fields. On August 18th, pelvic examination showed an increase in the size of the exudate in the right parametrium along with moderate tenderness and spasticity in the hypogastrium. The diagnosis of thrombophlebitis with multiple pulmonary infarcts secondary to parametritis and possible low grade pelvic peritonitis was made. The patient had received 3,200,000 units of penicillin and 2 pints of whole blood with no beneficial effect upon the course of the disease.

On August 19, 1946, a laparotomy was performed under spinal anesthesia through a left hypogastric paramedian incision. The left approach was used because her two previous operations were performed through right hypogastric paramedian incisions. On opening the abdomen there was an escape of a small amount of clear amber peritoneal fluid. The uterus appeared normal but both broad ligaments were markedly swollen. The pelvic veins were not palpated to avoid dislodging nonadherent thrombi. The posterior parietal peritoneum was incised over the bifurcation of the aorta and the inferior vena cava just

above the bifurcation was bluntly dissected free. No thrombus was found and it was ligated in continuity with a double strand of No. 3 chromic catgut. The incision in the peritoneum was closed with plain catgut sutures. The ovarian vessels were grossly free of thrombus and ligated *en masse* in the infundibulopelvic ligaments. The abdominal wall was closed in layers with No. 1 chromic catgut and silk to the skin. No significant cardiovascular changes were observed during the operation and the patient was returned to the ward with both lower extremities tightly bound with Ace bandages.

Her immediate postoperative course was stormy. On the fourth postoperative date there was an escape of serosanguineous fluid from the wound indicating a deep wound dehiscence. Her abdomen became markedly distended despite Miller-Abbott tube drainage and on the eleventh postoperative day she presented clinical evidence of small bowel obstruction. On August 30, 1946, under spinal anesthesia she was re-explored through the original wound. The deep layers of the wound were found widely separated. The small bowel was markedly distended down to the distal portion of the jejunum where an adhesion between two adjacent loops of bowel produced complete mechanical obstruction with volvulus of a short segment of bowel proximal to it. Numerous other strong adherent bands were found between the abdominal wall and the small

bowel. After lysis of all bands the relaxed loops were found to fill as the distended bowel emptied itself. The wound was closed with through-and-through interrupted wire sutures. The patient convalesced gradually and after a short bout of subacute obstruction on September 18th was discharged on September 30th as well.

Within two days after the primary operation of vein ligation, her pulmonary symptoms disappeared and her temperature returned to normal. The day following she developed marked vulvar edema which subsided within three days. Examination just prior to discharge from the hospital revealed slight edema of the ankles without supporting bandages, no gross evidence of superficial collateral venous circulation and no difficulty in walking. X-ray of her chest on discharge revealed completely normal lung fields.

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New Instruments

IMPROVED APPARATUS FOR SKELETAL TRACTION OF THE CERVICAL SPINE

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SINCE Crutchfield first used skeletal traction for the reduction of dislocated cervical vertebrae, it has gained wide acceptance and has marked the beginning of a more sane and successful concept of therapy. Prior to this time traction could be obtained only by the use of a halter, consisting of slings under the chin and the suboccipital region, which, of course, was uncomfortable. With this appliance the patient could rarely tolerate a traction force greater than 5 or 6 pounds which usually proved inadequate to effect the desired alignment. Furthermore, the halter type of apparatus was mechanically unsound. The resultant line of force was intermittently altered by motions of the mandible during eating, drinking, talking or yawning, frequently producing a seesaw type of motion at the site of fracture. Because of the mechanical limitations and its unpopularity with the patients, many surgeons advocated and practiced reduction of the dislocated cervical vertebrae by manipulation under general anesthesia followed by immediate application of an appropriate plaster cast. Just how many spinal cords were severed, contused, or further damaged during this procedure will never be known, but the mortality was so high during this period that a broken neck was almost synonymous with death.

In 1933, Crutchfield introduced the use of ice tongs which he applied to each parietal region. Although he was unable to effect the desired reduction of the cervical vertebrae, he was impressed by the comfort it afforded the patient. Later he modified the ice tongs to a smaller unit that could be placed in the upper eminences of the parietal region which enabled the patient to turn his head freely. By this method of securing skeletal traction he demonstrated by repeated roentgen observations that dislocations of the cervical vertebrae can be quickly and gently reduced by increasing the force. After reduction had been effected he was able to maintain alignment by the use of reduced force for as many weeks as indicated. One was indeed surprised how often the spinal cord was not irreparably injured and how rapidly its function frequently returned when the dislocated vertebrae had been gently and quickly realigned. Skeletal traction used wisely and patiently has eliminated all indications for manipulation of the cervical spine under general anesthesia in order to effect a reduction. Thus a new era of therapy was begun in 1933. Since then, the mortality resulting from dislocations and fracture-dislocations of this region has gradually decreased until now selected patients are considered amenable to treatment to the extent of complete rehabilitation. It is true that no apparatus is any better than the surgeon using it, but a mechanical device for securing traction of the cervical spine has commanded unusual interest because it has made possible a means of controlled therapy attended

by a decrease in mortality and an improvement of end results.

Neubeiser introduced a method of skeletal traction in 1933, which consisted of placing hooks beneath each zygomatic arch and using weights of 6 to 15 pounds. Zelmo, in 1939, reported the use of the same procedure with satisfactory results. Peyton, Hale and French, in 1944, reported the use since 1940 of the same method for securing skeletal traction. They had used maximum weights of 15 pounds and stated that the traction was comfortable and could be applied as long as desirable.

The placing of hooks beneath the zygomatic arches appears to be ingenious and simple but it does have the objection that the traction is produced in a plane anterior to that of the cervical spine. Therefore, patients might exist on whom this method would be contraindicated. McKenzie, in 1935, emphasized the limitation of halter traction and reported the use of ice tongs with a shoulder of $\frac{1}{8}$ inch from each tip and a locking device to keep the points fixed. Incisions of the scalp were required for their application. Cone and Turner, in 1937, reported the use of two burr holes in each parietal region which enabled them to tie a wire around the intervening bridge of bone in order to obtain traction. This procedure represents an operation of some magnitude. Barton, in 1938, reported the use of tongs made of rustless steel which required an incision of the scalp for application and which were held *in situ* by shoulders and a turn buckle. He was well satisfied with its performance in affording comfort and securing the desired results. Wright and Wunderly complained also of the inadequacy of the "Glisson sling." They devised an appliance which utilized the upper teeth and the suboccipital region for skeletal traction. Since it required the help of a dentist and considerable time to make the fixtures for the upper teeth, it is evident that this method does not lend itself readily or conveniently to the demands.

Recently the author described an apparatus for producing skeletal traction to the cervical spine which he devised and made while he was in a forward combat area in 1943. The principal material used was duraluminium obtained from the armor plates of destroyed American combat planes. Six separate units were used on twenty patients who were suffering with recent injuries to the upper spine. Once applied it did not require further adjusting. The maximum time that it was kept employed on any one patient was sixteen days. The maximum weight used for traction was 20 pounds although the usual weights were 10 to 16 pounds. In every instance the attending personnel were impressed by the comfort of the patient and the relative simple nursing care required. Since the earlier experience, an improved type of equipment has been used on six patients. The alteration consists principally of new stainless steel pins with points that will not penetrate the dura even if they should extend a few mm. beyond the inner table of the cranium. Furthermore, this portion of the pin has two flat borders so that traction against the bone of the skull may be transmitted by a flat surface and not by a cutting or round one. This renders the osseous density of the cranium capable of withstanding greater force transmitted through each pin. Also another stopguard has been added to permit the surgeon to have a choice. The larger stopguard is usually preferred for long flat heads while the smaller one is frequently used if the cranial contour is more oval.

A photograph and description of the parts and accessories of the apparatus are represented in Figure 1.

To attach the appliance to the cranium the surgeon and the assistant should wear sterile gloves but they may dispense with the usual scrubbing procedure. As the moving of a patient suffering with a fracture of the cervical spine entails considerable danger and discomfort, it is sometimes preferable to apply the apparatus wherever he may be and then move him while traction is being made. A sandbag is placed
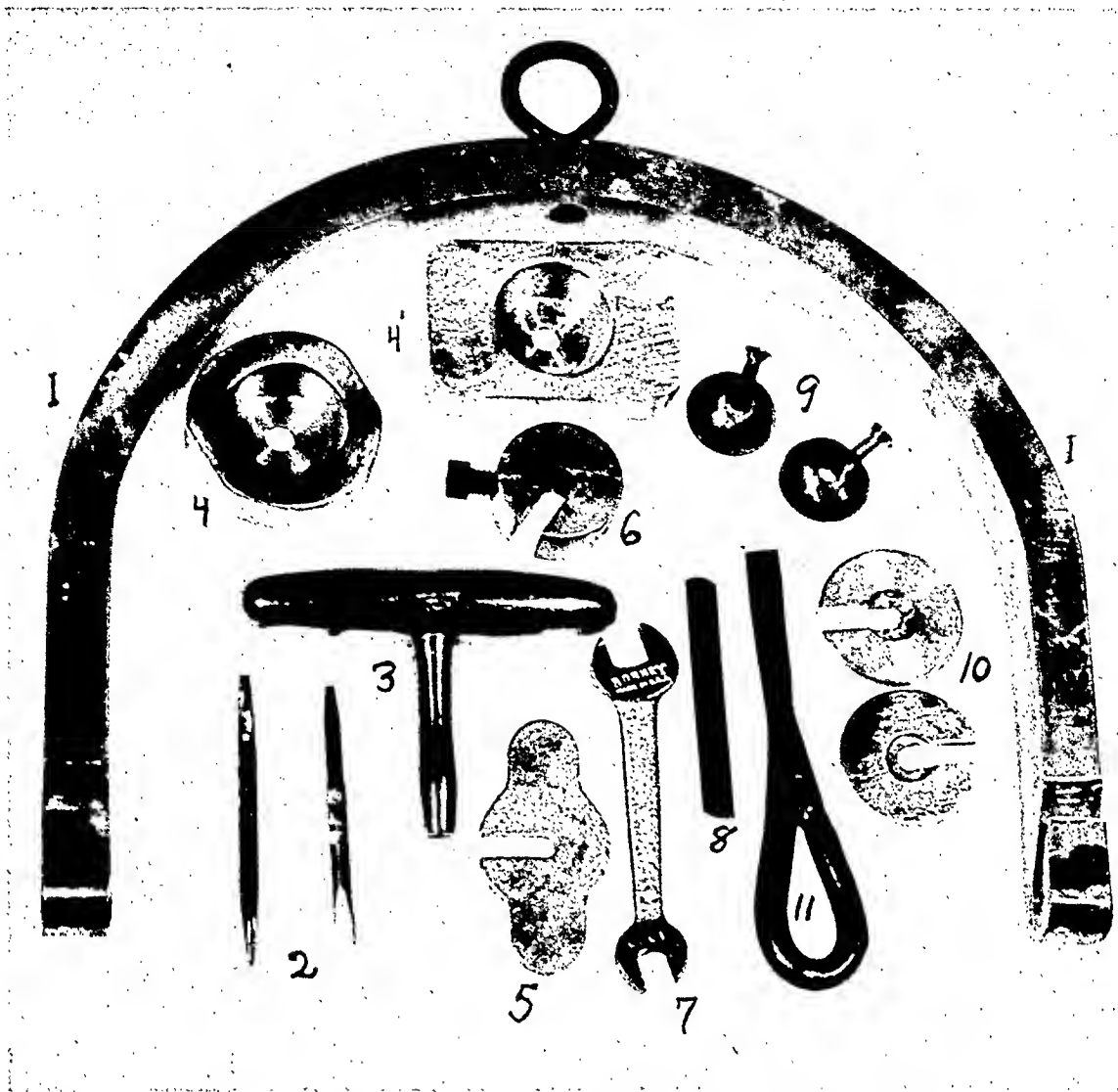
Fig. 1. Parts and accessories of the apparatus. (1) The duraluminium horseshoe frame has a width of 7 inches, a ring fastened to the toe and a slot in each heel. (2) Each stainless steel pin has a perforating type of point with bevelled shoulders recessed $\frac{3}{8}$ inch and a square head. There are three notches on the shaft to indicate the flat surface. The head is square to fit a T wrench. (3) T wrench (stock production, 4 & 44'). Choice of two stopguards made of duraluminium. Each has a concave base to fit over the scalp and a bore to allow for the wheal caused by an injection of procaine solution. (5) Depthmeter which is $\frac{1}{8}$ inch thick to be used for adults and $\frac{1}{16}$ inch thick to be used for children. (6) Guard with slot and set screw. (7) Wrench. (8) Ordinary glass file. (9) Hemispherical anchor with set screw. (10) Scalp plate with slot and concavity on one side. (11) Screw driver.

beneath the occiput, and an area of the scalp 4 cm. in diameter is shaved over each parietal eminence and cleansed according to a standard technic. At a point 3 cm. above and 1 cm. posterior to the pinna of the ear approximately 3 cc. of procaine solution is injected into the scalp and the pericranium on each side. The head of the pin is fitted to the T wrench, and the point is inserted through the stopguard, through the center of a piece of gauze 3 cm. in diameter, into the puncture wound of

the scalp made by the needle used for injecting the procaine solution, and passed through the soft tissues until the cranium is encountered. (Fig. 2.) The depthmeter and guard are now applied about the pin in that order. All are pressed firmly against the scalp while the guard is secured by the set screw using the wrench. By means of a file a mark is made on the pin for the purpose of detecting any slipping of the guard should it occur. The depthmeter is now removed and by rotation the pin is in-

serted into the cranium until the stopguard checks the progress. The three notches of the shaft should be left facing toward the dome of the scalp indicating that the flat surface of the point will bear the force of traction. The wrench, guard and stop-

desired weights are applied as shown in Figure 2.

The only hazard associated with apply-ing the apparatus is the possibility of in-serting the pin through the inner table of the cranium. This cannot happen unless the

INSERTING PIN

a
Depthmeter ⅛" thick between guard and guard stop.

b
Depthmeter removed allowing pin to enter cranium ⅛".

APPARATUS APPLIED

Skull plate

Hemispherical anchor
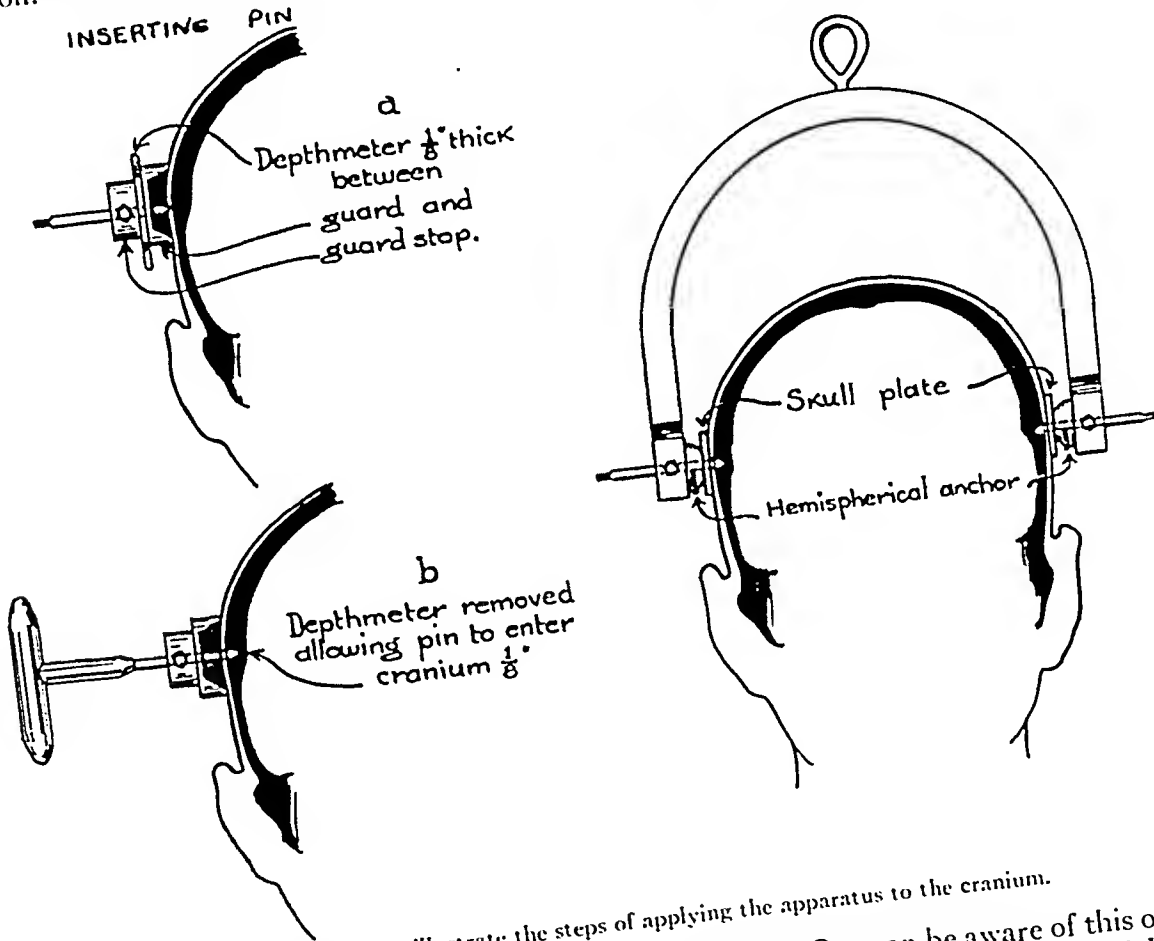
FIG. 2. Drawing to illustrate the steps of applying the apparatus to the cranium.

guard are removed, the hemispherical anchor threaded over the pin with its con-vexity toward the scalp and the assistant holds the pin in situ until the procedure is repeated on the other side. The slots of the duraluminium horseshoe frame are then placed about the pins and secured firmly by the set screws. The scalp plate is placed against the gauze and the hemispherical anchor is slid into its concavity forming a universal joint, thus permitting the plate to lie firmly against the scalp regardless of the shape of the skull. A rope is tied to the ring, led over an adjustable pulley and the

guard slips. One can be aware of this occur-rence by observing the mark which has been made on the shaft. Furthermore, when the recessed shoulders strike the outer table of the cranium it is very difficult to force the pin deeper. Even if the pin should penetrate the inner table for a few mm., the point is so designed that the dura will be depressed and not penetrated as demon-strated on a cadaver. A little experience will convince one that this possible hazard requires only moderate caution and it should not constitute a fear. If the patient is under fourteen years of age, the depth-
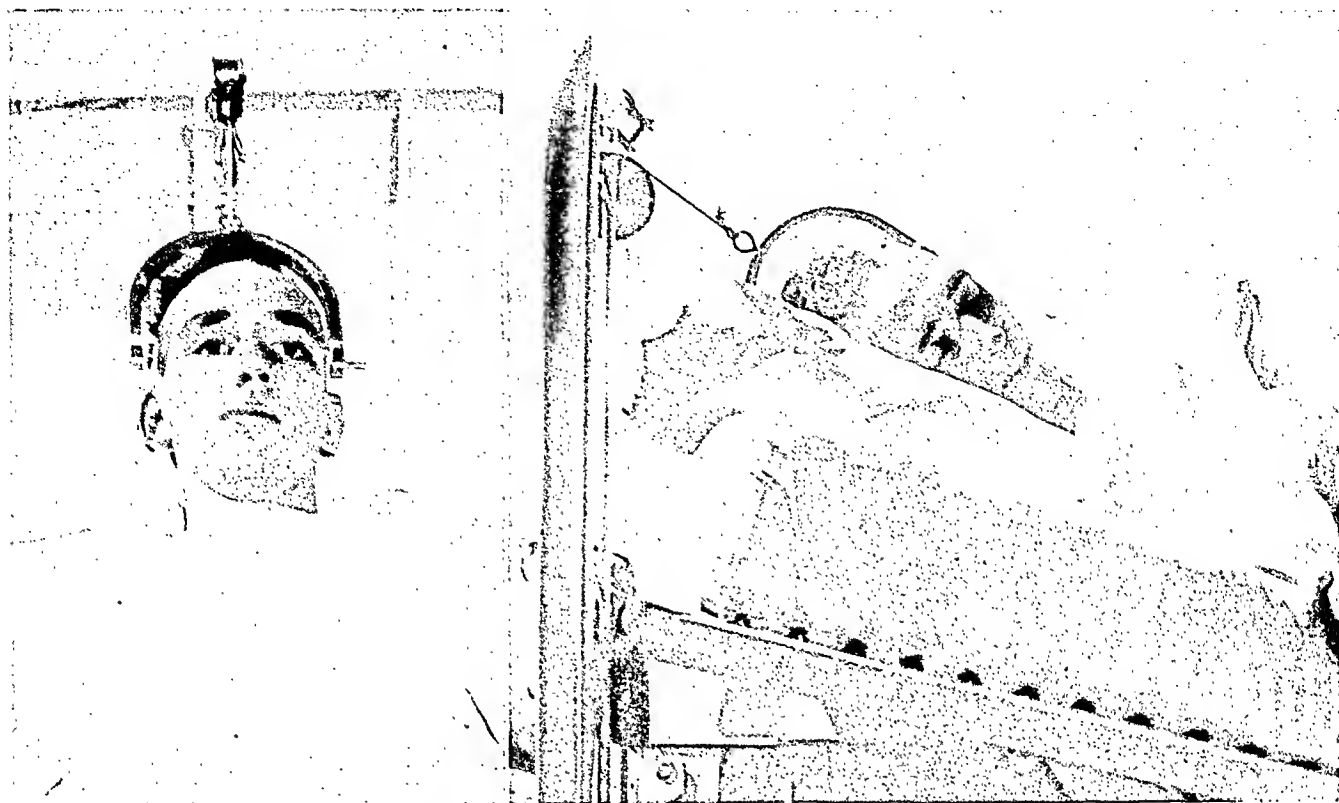
FIG. 3. Apparatus in use. A, patient in supine position; B, patient lying on right side.

FIG. 4. A, anterior dislocation of first and second cervical vertebrae on the third with right pedicles locked; six weeks' duration. B, reduction and alignment effected by skeletal traction using 35 pounds force.

meter of $\frac{1}{16}$ inch thickness should be used rather than that of $\frac{1}{8}$ inch. Figures 3A and B show the apparatus in use. In this particular instance the pulley is stationary and the adjustments have been made by raising or lowering the mattress frame which also affords a method of countertraction.

One of the six patients treated by this revised apparatus had been injured about six weeks previously. There was an anterior dislocation of the first and second cervical vertebrae onto the third with the right pedicles locked. The apparatus was applied and a traction of 20 pounds proved inadequate to effect reduction. The weights were increased until 35 pounds were used for six hours on each of two consecutive days during which time the patient was watched carefully and x-ray studies were made at frequent intervals. After the first two hours of each day he complained of tired, aching pains in the cervical region that required codeine and aspirin. When unlocking of the pedicles had been accomplished, the weights were reduced to 12 pounds; they were kept applied for thirty days. (Figs. 4A and B.) The patient spent the remainder of his convalescence in comfort. He stated that he was unaware that there was any traction on his neck except at times when he would forget himself and attempt to sit up. When the patient was ready for a plaster cast, the apparatus was detached simply by loosening the set screw of each slot of the horseshoe frame. A small piece of gauze saturated with colloidon applied to the resulting puncture wounds of the scalp afforded adequate surgical dressings.

It appears that traction to the cervical spine afforded by the various appliances attached to the cranium is more or less identical. All fixtures have withstood a force in the plane of the cervical spine adequate to accomplish the purpose. Therefore, the apparatus of choice would depend upon its familiarity to the surgeon, the comfort to the patient, the ease of application, the lack of hazard to the intracranial contents, the security of the attachment and the amount of adjusting required while it is kept *in situ.*

The horseshoe type of apparatus with little variation (Fig. 1) has been used on twenty-six patients. After the first few hours it has been comfortable. It has fulfilled the requirements of the criteria mentioned, to the extent that it may have earned consideration for its adoption when the surgeon is confronted with the particular problem for which it has been devised and made.

CONCLUSION

A horseshoe type of apparatus for attachment to the cranium to afford skeletal traction of the cervical spine has been described and used satisfactorily on twenty-six patients. It has been comfortable, required no incisions of the scalp, remained firmly attached to the cranium without further adjusting and produced no injury to the intracranial contents.

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TRACHEAL AIRWAY FOR USE DURING TOTAL LARYNGECTOMY*

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DURING performance of total laryngectomy when the trachea has been transected just below the cricoid cartilage, it is desirable that the distal tracheal stump be held out of the way so that no blood enters the tracheobronchial
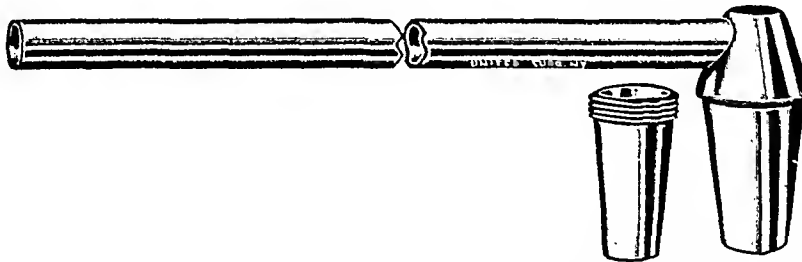
FIG. 1. Breathing tube for laryngectomy which consists of a tapered angled tube with two detachable ends (large and small). A useful devise to hold the tracheal stump out of the way so that blood will not enter the tracheobronchial tree.
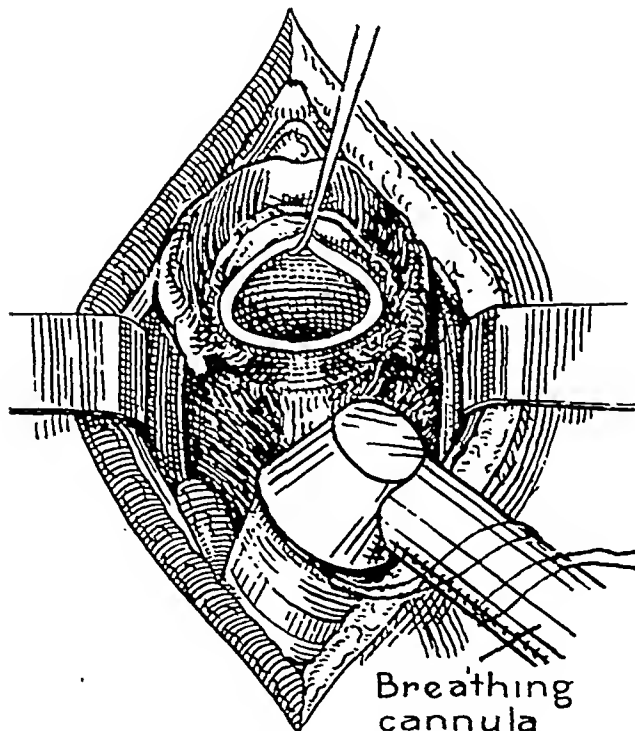
Breathing
cannula

FIG. 2. Diagram showing tube in place during laryngectomy. The trachea is cut across just above its upper ring and the breathing cannula is inserted into the open end of the trachea. Two heavy sutures are passed through the tracheal wall and wrapped around the angled tube to keep it in place.

tree. This phase of the operation may be particularly troublesome to the patient and annoying to the surgeon, especially when operating on individuals with bleeding tendencies. If the tracheal stump can be plugged with a tapered tube which is bent at such an angle so as not to be in the way of the operator, the safety factor of the surgical procedure is increased and no unnecessary time consumed.

The device shown* in the accompanying diagram consists of a tapered angled tube and is provided with two detachable ends of different sizes, large and small. (Fig. 1.) The tube is held in place by passing a heavy suture through the stump and winding it several times around the tube. (Fig. 2.)

* This instrument is manufactured by and may be purchased from the United Surgical Supply Company

G. Gocaa reports two instances of tuberculosis of the thyroid both proven histologically. Both patients also had tuberculous lesions elsewhere. In one, surgery was contraindicated because of advanced osseous tuberculosis and the "acuteness" of the degree of the perithyroid tuberculosis infiltration that was also present. The other woman had a cold abscess in an adenoma of the thyroid, but her general condition was better and her pulmonary tuberculosis was healed. Hence, a partial thyroidectomy was done in her case. (*Richard A. Leonardo, M.D.*)

Editorials

SURGERY AND PSYCHOSOMATIC DISEASE

DURING World War II the medical profession became more acutely aware of psychosomatic disease than it had been for many years. However, this is a matter of terminology rather than actuality for the practitioners of medicine for generations have recognized that somatic pain may have its origin in psychic conflicts. That these psychosomatic phenomena may simulate surgical disease is not at all surprising when one recognizes the protean nature of the symptoms of so many of the surgical diseases and how few are the objective manifestations.

Abdominal diseases are expressed in the form of pain, alterations in the normal gastrointestinal and urinary responses such as anorexia, nausea and frequency of urination. The objective findings are also very few in the form of fever, tenderness and splinting of the musculature. With the possible exception of fever all of these may be present as expressions of psychosomatic disease. Fever may be present in minor degrees at the time of ovulation in the female and for a variety of minor causes including the psychic stress attending hospitalization. Selective dyspepsias may be an aid in the diagnosis of cholecystic disease yet this also is not infrequent in patients with psychosomatic disease.

How then may the organic disease be differentiated from the functional? Certainly all will agree that the operative process of elimination is neither the ideal nor even often warranted. This is the process of exploratory laparotomy, or the appendectomy or cholecystectomy for chronic but undiagnosed pain. Can the psychiatrist answer the question; can he differentiate the right lower quadrant pain of psychosomatic origin from that of the recurrent inflammations of the appendix? No! The psychiatrist can locate underlying causes for psychic conflicts within the patient but usually he requires that the clinician eliminate organic causes before he begins. Psychic conflicts of some kind are undoubtedly present in all humans to a greater or lesser degree and can be elicited by careful interrogation. That these are present does not mean that they are the cause of symptoms. One must beware of the adage *post hoc ergo propter hoc*. The psychosomatic patient may also have acute appendicitis, chronic cholecystitis, carcinoma of the pancreas and even carcinoma of the breast. Almost every patient who dies of carcinoma of the body or tail of the pancreas has been diagnosed as a psychoneurotic.

Wherein lies the answer to this double-edged sword of the term psychosomatic disease? Now, as it has for previous medical generations, it still remains a problem for the conscientious investigation by the

clinician, namely, careful analysis of the pain, its character, timing and radiation; a painstaking review of the complete anamnesis, again stressing the sequence or timing of the symptoms; a complete physical examination, utilizing all of the art of this procedure as distraction and false suggestion.

These, coupled with all of the refinements of scientific adjuncts, laboratory tests and radiologic studies, will bring to light most of the organic disorders. Discussion of the special problems with colleagues in the fields of internal medicine will further aid in the winnowing of the organic from the functional.

Yet, when all means available for study have been exhausted, there will still be patients in whom a failure of diagnosis must be admitted. Is the surgeon justified in considering his exploratory laparotomy a diagnostic procedure? This is an especially difficult question in a patient whose emotional instability is quite obvious. To operate upon such a patient will inflict real and perhaps irremediable psychic trauma to that patient. To overlook an organic lesion will condemn that patient to a life of continued suffering if not worse. Every surgeon of experience can recollect innumerable instances when he has been faced with this question. At present we have no answer. The experience and acumen of the surgeon, the maturity of his reasoning must be called into play. If he errs, he will have a sick and dissatisfied patient and he may even be the subject of implied ridicule of his fellows.

Every surgeon is faced with this problem. So we must be tolerant when we know that others are faced with similar problems. When we see patients who have the same symptoms after a cholecystectomy as before, we realize that one of our fellow surgeons faced this problem and decided it according to his best judgment. Not alone that, but now we must again study this patient from every angle to make sure again that there is not some organic disease before we label them as psychoneurotic.

The diagnosis of a psychosomatic disorder can be made only after all of the studies that can be marshalled to aid in diagnosis have been exhausted. And then we must be especially alert, lest we ascribe any real syndrome, that they may subsequently develop to their psychoneuroses.

WILLIAM C. BECK, M.D.

THE SHIFTING ECONOMIC SITUATION AS A DETERRANT TO THE EDUCATION OF THE SURGEON*

ALL those interested in the future education of the American surgeon must study and evaluate the changes which modern economic situations have made in hospital practice. Twenty years ago a great many of our largest hospitals had a major number of so-called charity or free beds frequented by the poorer class where such people who could not afford the services of a private surgeon went to have their surgery performed. The teachers of surgery in the great schools were very jealous of their prerogatives as regards these "open" or charity beds because this was the material which they could utilize as they saw best in the final education of the young surgeon.

The education of the surgeon and the physician differ essentially, in that although many may profit from the examination of a patient with a cardiac murmur or some unusual medical sign, only the person who himself conducts the operative procedure can fully benefit from that experience. This handicraft aspect of surgery is something that must be learned and it must be learned under the most careful tutelage. After several years as an intern or assistant resident surgeon or fellow, the young surgeon reaches the point where he cannot progress further unless he has individual surgical responsibility and the opportunity to operate independently and by himself. The experience acquired by such operations gives to the young surgeon confidence and practice which are essential to his final development.

Shifting economic trends now are jeopardizing the existence of so-called charity or "open" beds in many of our greatest hospitals, either because the people are in large part becoming insured through group insurance systems, or because industry is taking over the care of its laborers and has its own doctors to look after them. Such patients are now private patients in the sense that a contract exists which places them under the care of specific individuals and their surgical care cannot be turned over to other than those mentioned in the contract. The number of "open" or charity beds is further diminished by the rising income of all workers which has now reached the level at which most hospitals could fill a greater percentage of their beds with paying patients. Hospital trustees, impoverished by the rising cost of hospitalization, have noted this change in the economical status of their patients and are anxious to close as many of their public beds as possible and thus cut down their mounting deficits. There can be no question but that the shift toward insurance schemes, whether public or private, is sure to continue and to advance. Whether or not it can survive a depression will depend in great part on the amount and vigor of Government aid.

Irrespective of future alterations in the economic situation, the present status must be studied and those interested in a source of material to fully train the surgeon must modify their plans for as teachers it is their responsibility to see that this important matter continues. It would appear that hospital staffs, including the teaching staffs of university hospitals, in order to cover this might organize themselves into some kind of "group practice," including in their group the senior members of the house staff so that when the hospital in which they work takes on people who are insured, whether by contract with a labor group, a bank or a business organization, it would be equally satisfactory to the insured if the resident surgeon, as the right hand

man of the chief of staff, be also in the group allowed to perform the final surgical ordeal. Some such organization must be found shortly for the economic situation is affecting not only this vitally important matter of the training of the surgeon, but the education of medical students as well. The sooner the teaching profession wrestles with this problem the sooner it will be accomplished. I have no doubt that those people desiring to be insured will accept any agreements which the leading teachers wish to bring forward.

This is written only to stimulate those responsible to make such studies now and to plan carefully their educational program in the future, basing it on the utilization of insured or semiprivate cases rather than on the continuously disappearing supply of so-called charity or "open" beds.

ELLIOTT C. CUTLER, M.D.

STUDENTS who watch operations have never really been able to see or learn everything which goes on during the operation because they are usually too far away from the surgeon. To overcome this Trimble and Reese introduced television in an operating room at the Johns Hopkins Hospital, with excellent results. Even the most minute surgical procedure can now be made plain to the observer, surely a worthy advance in the teaching of operative surgery. The day is not far removed when all-important teaching centers will have at least one of their operating rooms, and its associated amphitheater, so equipped. (*Richard A. Leonardo*, M. D.)

POSTERIOR SPHINCTEROTOMY

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AN incision of the subcutaneous portion of the external sphincter is correctly called a sphincterotomy. Such a procedure was first advocated by Boyer back in 1788[1] for the cure of fissure in ano. It is not definitely known whether or not he recommended division of the sphincter through the base of the fissure but Earle in 1911[1] did recommend it on the theory that it was the spasm that caused the fissure and deep incision would obliterate this spasm. As approximately 90 per cent of fissures occur at the posterior commissure his incision must have been made in the latter location most of the time. Since then, other operators have considered it more logical to make the incision in one of the lateral quadrants rather than either in the base of the fissure or at the posterior commissure.

An incision of the pecten band, on the other hand, is correctly called a pectenotomy. It was first advocated by Miles[2] in 1918 who used it not only for the cure of fissure in ano but also as an integral step in his hemorrhoidectomy operation. He divided this band in the right posterior quadrant.

Gradually in this country a group of proctologists have readopted the custom of making sphincterotomy in the posterior midline[3-5,22] and their operation, often of necessity as well as by intent, has also included incision of the pecten band whenever present. In other words, they perform simultaneously both a sphincterotomy and a pectenotomy located at the posterior

commissure. The reasons will become clear later on in the text.

Confusion as to the difference between sphincterotomy and pectenotomy still exists. However, this confusion disappears whenever a clearcut conception of the normal anal musculature is contrasted with the pathologic condition inherent in pectenosis. Both of these subjects can stand repeated exposition.

ANATOMY OF THE ANAL MUSCULATURE

It is only within the last few years that accurate information concerning the normal anal musculature was established by Milligan and Morgan,[6] Blaisdel,[3] Levy[7] and Gorsch.[8] Certain salient features of this mechanism should be emphasized as follows:

1. The external sphincter is composed of three divisions with the two deeper portions (i.e., the superficialis and the profundus) surrounding the internal sphincters and, contrarily, the subcutaneous portion being distal or caudal to and in the same vertical plane as the internal sphincter. (Fig. 1.)

2. The presence of and the function of the conjoined longitudinal muscle should not be overlooked. This muscle is a continuation of and a confluence of the longitudinal bands on the outside of the large intestine plus some of the fibers of the levator ani. It surrounds the internal sphincter, encases the bundles of the external sphincter and becomes inserted in part into the skin of the anus at Hilton's white line in the intermuscular
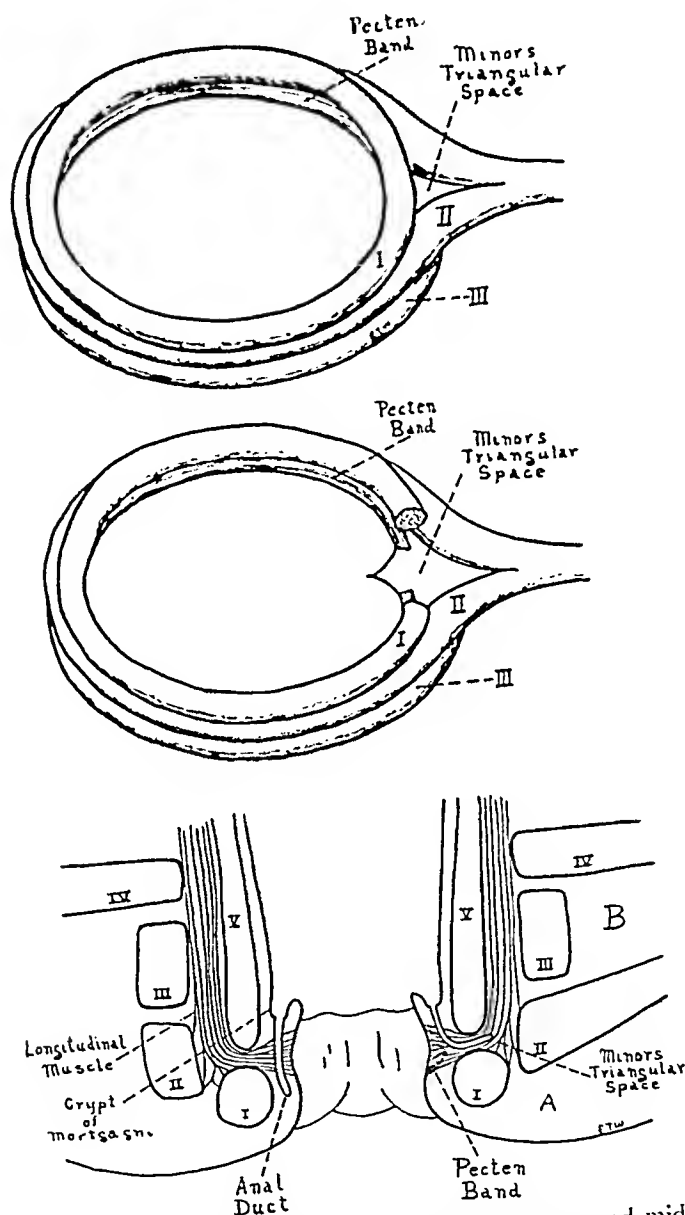
FIG. 1. Diagram illustrating the anatomy of the anal sphincters. The upper and middle plates depict the three portions of the external sphincter looking from without inward through the anus. The lower plate depicts a sagittal section through the anus and sphincters. In all three plates I represents the subcutaneous portion of the external sphincter; II represents the superficial or superficialis portion; III is the deep or profundus portion; IV is the levator ani muscle while V is the internal sphincter. The upper plate shows the doughnut-like character of the subcutaneous and deep portions of the external sphincter and the way the tangential legs of the superficial portion go off to produce the anococcygeal attachment with Minor's triangular space between the two legs. The middle plate shows how the latter space is opened up by a posterior sphincterotomy. The lower plate shows the "directive" influence of the longitudinal muscle on the anal glands both laterally (left) and at the posterior commissure (right). It should be noted that there is a distinct difference between an anal duct (called a "crypt" when infected) and the crypt of Morgagni. In this plate the anal duct or "crypt" is pictured opening into the crypt of Morgagni as it does in the human anus. Spaces A and B are the superficial and deep post-anal spaces, respectively, with which Minor's triangular space communicates. The location and strangulating influence of a pecten band is clearly seen.

septum.[4–6,8,22] (Fig. 1.) This muscle becomes more fibromuscular in character the nearer it approaches the skin of the anus and the latter characteristic is an important factor in the production of some of the pathologic conditions to be described. As Gorsch says, "these longitudinal fibers are of special importance on account of their directive influence on the extension of infectious processes from the pecten and the rectum to the perianal tissues."[8]

3. There are several perianal spaces which, although they are very important in the surgery of this region, have seldom been given the notice they deserve. The most important one is the actual or potential space called Minor's[9] triangular space lying between the two tangential legs of the superficial portion of the external sphincter as the latter go off to become attached to the coccyx. (Fig. 1.) For purposes of simplification it may be stated that the deep and subcutaneous portions of the external sphincter form doughnut-like rings above and below this space, respectively. Technically, the latter should be called the posterior intermural space to differentiate it from the superficial and deep postanal spaces which are below and above the tangential legs of the superficial external sphincter,[8] respectively, and with which it potentially communicates. (Fig. 1.) This potential communication is posterior to and outside the two doughnut-like deep and subcutaneous portions of the external sphincter. In addition to these three spaces there are of course the two ischiorectal spaces on each side of the anus and rectum bounded above by the levator ani muscles, by the skin below and by the pelvis and rectum on either side.

PECTENOSIS

A pecten band (pectenosis), on the other hand, is a pathologic structure. It was first mentioned and given its name by Miles[2] in 1918 but was studied and described in greater detail by Abel[10] in 1932. It pertains to a fibrotic band occurring in

and beneath the transitional skin of the anus in the region of the intermuscular septum. The etiology of this condition had been variously laid at the door of both chronic passive congestion and chronic infection. However, when the classical work of Tucker and Hellwig[11] confirmed by Hill, Shryock and Rebell[20] put onto a firm foundation the presence of some infected anal ducts ("crypts") beneath the skin of the anus, it was possible to incriminate these latter structures as being the prime causative agents. Certainly they can be demonstrated to exist coincidentally with a pecten band in practically all cases whenever the latter is found to be present. Apparently the fibromuscular insertions of the longitudinal muscle are transformed into fibrous insertions in the presence of and as a result of chronic infection existing within and around these anal ducts,[5] and gradually the intermuscular septum becomes filled with and replaced by a band of pearly white connective tissue.[22]

Contraction of this fibrosis produces several pathologic effects. First, it strangulates the neck of the anal ducts as the latter pass between the fibrotic fibers, turning the ducts into little undrained abscesses which have a tendency to burrow and thereby produce most of the various perianal and perirectal abscesses, blind fistulas, complete fistulas and fissures. Second, in due course of time this dense contracted band of fibrosis within the anus prevents proper relaxation of the sphincters. This can proceed to such an extent in elderly patients that the anus actually becomes a stricture. Third, it either anchors the skin inside the anus onto itself or turns it into a fibrous type of tissue which is unelastic, avascular, splits easily and gives rise to constant discomfort.[12,22]

Therefore, the finding of fissures, abscesses, posterior fistulas, pecten bands and even chronically sore "behinds" usually infers the presence of some acute or chronically infected crypts.[3,5,11,12,15–20,29–31]

It might be argued that whenever a pecten band is encountered, simple pecte-

notomy would be all that was necessary to sever it and lay open to drainage the underlying infection but this does not take into account the anatomy of the sphincter at the posterior commissure. As we have shown[5] infected anal ducts in the latter location have a tendency to be "directed" by the longitudinal muscle into Minor's triangular space and it requires not only a pectenotomy but also a posterior sphincterotomy to adequately drain this space.[9]

INDICATIONS FOR POSTERIOR SPHINCTEROTOMY

Inference as to the indications for sphincterotomy located at the posterior commissure has been made in the preceding sections but for those not versed in proctology a more detailed exposition will follow. We personally have performed such a procedure in over 800 patients either in connection with the several conditions mentioned in the previous and following sections or as we found that it had secondary advantages when done in addition to some proctologic operation. Our experience has shown it to be indicated in the following conditions: (1) cryptitis; (2) strictures about the anus (3) posterior perianal abscesses; (4) complete fistulas at the posterior commissure; (5) blind fistulas at the posterior commissure; (6) posterior fissure in ano; (7) hemorrhoidectomies; (8) certain cases of pruritus ani; (9) for operative intrarectal exposure.

Prior to taking up these items in more detail we wish to emphasize that whenever the term posterior sphincterotomy is used henceforth in this article, it will be assumed that pectenotomy is included if pectenosis is present.

Cryptitis. When it is more generally realized how important these infected anal ducts or crypts are in the production of so much anal and perianal pathologic conditions, a search for their presence will be more frequently made and the cryptectomy operation will become a more common procedure. Our conception of a crypt is

simply that of an anal duct which was originally of microscopic size but which became enlarged and elongated due to repeated bouts of infection. During the course of this process and consequent thereto pectenosis or a pecten band was produced which partially or completely strangulated the neck of the crypt thereby obliterating adequate drainage and giving rise to further elongation and enlargement as well as symptoms.

The term crypt is used very loosely and is surrounded by much confusion in the literature. This is because there are two crypts—the crypts of Morgagni and the aforementioned infected anal ducts commonly called crypts. The crypts of Morgagni are simply the cup-like depressions to be found behind the mucocutaneous or pectinate line in the bases of the sulci between the columns of Morgagni. They are wide open, shallow depressions of varying but minor depth and it is hard to conceive how they could burrow their way down behind the pecten band which is distinctly below or distal to them. The anal ducts on the contrary have their openings in the crypts of Morgagni but extend therefrom downward through the fibers of the longitudinal muscle or pecten and into or among the various members of the anal sphincter. In the lower animals they are present in greater numbers and exist as definite secretory glands. After they become infected they are generally spoken of as crypts. This terminology has come about through popular usage and arises from the fact that the terms crypt and cryptitis are shorter and more easily enunciated than anal duct and anal ductitis and because they were formerly thought to be the crypts of Morgagni. If this difference is kept in mind, all confusion disappears. To repeat, the cup-like depressions between the columns of Morgagni are the crypts of Morgagni while a crypt on the other hand is an *infected* anal duct which empties into the crypt of Morgagni. As previously stated this conception is based on the pathologic studies made by Tucker and

Hellwig,[11] Hill S. Shryock and ReBell[20] and others.[5]

The cardinal symptoms of cryptitis are burning, irritation or aching plus periodic attacks of anal soreness. Crypts can only be demonstrated by means of a crypt hook. Therefore, let those critics who do not own such a hook or who do not use it make no doubtful or disparaging remarks concerning the presence of these pathologic structures especially in the light of the vast amount of literature concerning them which has appeared in the past few years.[3, 5,11,12,15—22,25—31] Furthermore, although the recognition of their importance in the production of proctologic pathologic conditions may be a moderately recent development, nevertheless their presence in the adult human anus was known and recorded back in 1732 by Winslow.[23]

Generally whenever one crypt is found, there are usually at least two more and always one of these is at the posterior commissure and more often than not the latter one has its distal tip pointing into Minor's triangular space.[5] (Fig. 1.) We used to excise the one offending crypt in the clinic or office in an ambulatory manner[19] but so often we found that while we may have eradicated one of the crypts a second one and even a third one was lighted up by the procedure, consequently more operations became necessary much to the patient's annoyance. Furthermore, a crypt at the posterior commissure with its tip out in Minor's triangular space was not always eradicated by such a simple procedure. Consequently, we now put these people into the hospital and do a thorough, multiple cryptectomy plus posterior sphincterotomy.

Furthermore, not too rarely a patient will be seen who gives a history wholly consistent with that of fissure in ano but no fissure can be found. However, at the posterior commissure is found an area of exquisite tenderness, induration, a pecten band and even spasm of the sphincter. The crypt hook can be pushed down through a cryptic opening, beneath the pecten band and into the area of induration. Excision of all of the tissues on the hook plus posterior sphincterotomy will affect a cure. Waiting for nature to take its own course leads to either a fissure or a fistula. Also, not too rarely, the proctologist meets a patient who has had a hemorrhoidectomy unsuccessfully performed for the relief of symptoms consistent with cryptitis. Examination reveals the offending crypt still at the posterior commissure but now also ensnared by the fibrosis produced by the hemorrhoidectomy. Cryptectomy plus a posterior sphincterotomy gives permanent relief.

Strictures about the Anus. As we and others have pointed out many a case of long-standing, contracted anus is the result of this chronic infection of the anal ducts or cryptitis. However, other causes for anal and lower rectal stricture occur such as past or recurrent abscess formation, chronic fistulas, false appositions following hemorrhoidectomies, lymphogranuloma venereum, congenital malformations, etc. Whatever the cause for a stricture in this location, it will be found that a posterior sphincterotomy not only increases the size of the anus but lays it open long enough to allow for resolution of a great deal of the fibrosis and to give time for epithelialization to take place over the ends of the severed stricture thereby preventing premature reapposition. When the stricture is somewhat above the anus, as in chronic abscess formation for instance, it can be incised as a part of a posterior sphincterotomy and then after control of bleeding the superior edge of the mucous membrane can be brought down and anchored into the base of the wound at the level of the internal sphincter or erstwhile pectinate line.[24]

Posterior Perianal Abscesses. Perianal abscesses near the posterior commissure practically always have an internal opening in a crypt.[15—18,22,25,27,29,30,31,33] The latter is usually at the posterior commissure. Its opening became strangulated by the fibrosis of the longitudinal muscle fibers[5] and

the infected stricture burrowed its way either down between the mucous membrane and the external sphincter or out through Minor's triangular space into the superficial postanal space and thence into the perianal tissues. (Fig. 2, middle plate.)

considered best by most operators to simply incise the abscess and await the subsequent production of the fistula. Incision or excision of such a fistula will usually require total incision of the subcutaneous portion of the external sphincter at the
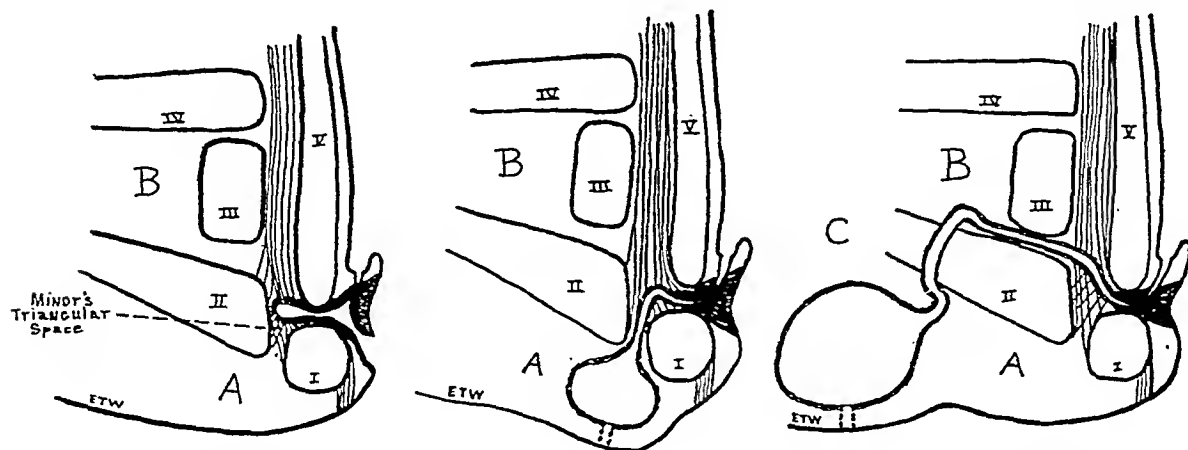
Fig. 2. Diagrams illustrating the varying progress of a strangulated crypt. All plates represent sagittal sections through the posterior commisure. The left one depicts the production of a fissure-in-ano (so-called). Really it is an intra-anal fistula; a crypt became infected, produced a pecten band, was strangulated by the pecten band, burrowed back toward or into Minor's triangular space and finally ruptured out into the anus lower down. The middle illustration is that of a post-anal abscess and fistula with the crypt making its exit from Minor's triangular space upward into the deep post-anal space and thence to the right or left into the ischiorectal space.

Following incision of the abscess, a crypt hook can often be introduced into the internal cryptic opening and forced down through the pecten band and on into the abscess cavity. Incision on the hook is made and is followed by a full posterior sphincterotomy taking off as much skin from the other buttock as has been laid open on the affected side. (Fig. 3.) A permanent cure is had and a secondary operation for a fistula is eliminated. No deformity and hardly any scar will follow.

Complete Fistulas at the Posterior Commissure. The vast majority of ischiorectal abscesses have an internal opening in a crypt.[33] When this crypt is at the posterior commissure, it has a tract running down behind the pecten band, backward through Minor's triangular space and then either upward into the deep postanal space and thence into the ischiorectal space or downward into the superficial postanal space and thence into the latter space. (Fig. 2.) Such tortuosity is too much for any unyielding probe; consequently, it is

posterior commissure. This should include the severing of all of the pectinotic fibers in order to secure a smooth, undeformed and elastic anus afterward.

Submucous fistulas and abscesses in this location should also have the benefit of more adequate drainage and obliteration by means of sphincterotomy and pectenotomy after the incision of the tract higher up has been completed.

Blind Fistulas. Not too uncommonly a blind fistula is found at the posterior commissure. Its one opening is usually at the level of the pecten band and allows only partial drainage either into the anus or into the rectum or both. The cavity behind the pecten band may be small or large but the production of seropurulent discharge is often voluminous. For both exploration and cure a right angled probe is necessary. (Fig. 3.) Here again it should be stated that following its eradication the remaining fibers of the pecten band should be incised in order to obtain a smooth, elastic anus once more.

FIG. 3. Illustrating the cure of a blind fistula at the posterior commissure. A, a properly made right angled probe with a speculum inserted into the anus; B, the hook has been introduced into the fistula, the speculum has been withdrawn and the scalpel is incising down onto the hook; C, the wall of the fistula has been excised and a posterior sphincterotomy plus a pectenotomy have been completed. D, shows how much additional skin must be excised from either side in order to delay healing until complete granulation has taken place. The resulting scar will be in the base of the commissure and will be hard to find within three months.

Fissure in Ano. In the opinion of several American proctologists sphincterotomy, or at least part of it (pectenotomy), is a "must" in fissure work.[3-5,9,10,12,22] Both theoretically and practically it fills the need for a procedure which will definitely cure and as well definitely prevent the frequent recurrences which so often follow less radical procedures, such as simple excision, dilatations, cauterization, injections, etc. Sometimes the use of a right angled probe shows the fissure to be a true blind fistula going back into Minor's triangular space.[5] Such a finding confirms the reasoning that a fissure is simply an infected crypt which has ruptured through the anal wall. (Fig. 2.) The fact that 95 per cent of fissures occur at the posterior commissure means something and this something has to do with cryptitis plus the difference in the anatomy of the sphincters at the posterior commissure. As previously described crypts in the latter location have their ducts directed backward by the longitudinal muscle toward and into Minor's triangular space and a pecten band turns

any infection in the duct into an intermural abscess. (Fig. 2, left plate.) During infection this little abscess is compressed and extension of its contents is brought about. This extension follows the line of least resistance and in fissure cases this line appears to be through the thin intra-anal skin just below the pecten band. Therefore, a fissure usually should be considered and treated as being a fistula with its primary opening in the original mouth of the crypt up in the rectum and its secondary opening below the intermuscular septum. The tract meanwhile traverses backward behind the pecten band toward if not into Minor's triangular space. (Fig. 2.) Cure requires excision of the whole tract, plus drainage of Minor's triangular space plus time enough for granulation to take place before full re-epithelialization occurs. Posterior sphincterotomy accomplishes all of this.

Hemorrhoidectomies. There is one school of thought headed by Miles and Abel which considers pectenosis to be the result of chronic passive congestion accompanying or following hemorrhoids. Careful and prolonged observations of over a thousand cases of hemorrhoids has convinced us that the majority of hemorrhoids are not accompanied by pectenosis except when there is also a coincidental cryptitis. However the case may be, some proctologists advocate routine posterior sphincterotomy in hemorrhoidectomies. We go further. We believe it to be a must in this operation[14] for several reasons: (1) It tends to prevent false re-appositions and thus stricture formation; (2) it definitely diminishes postoperative edema plus the pain and discomfort that accompanies the latter; (3) it abolishes spasm of the external sphincter allowing earlier, freer and more comfortable bowel movement; (4) postoperative skin tabs are less likely to form; (5) a simple pressure dressing applied into the resulting wide open anus prevents any oozing from those tissues which are distal to the transfixed hemorrhoidal pedicles.

Elimination of postoperative edema alone is an adequate indication for its routine use in hemorrhoidectomies for this edema is probably the greatest factor in the production of disability, prolonged hospitalization and morbidity following hemorrhoidectomy operation.

Pruritus Ani.[10] Spiesman,[13] J. W. Morgan[12] and others have advocated pectenotomy for pruritus ani. Gorsch finds posterior sphincterotomy theoretically indicated in cases accompanied by cryptitis but does not wax enthusiastic about it. We have not had much success with it in pruritus ani when performed on an empirical basis. However, when a case is encountered which has undergone irreversible changes in the form of large folds of hyertrophied skin alternating with deep creases, no relief and certainly no cure can be had until the skin is smoothed out. Excision of the folds, undercutting of the remaining skin strips and deep, wide posterior sphincterotomy followed by frequent digital prevention of too rapid healing results in a smooth, re-epithelialized perianus which has the form of an inverted funnel. Treatment for the pruritus then has some chance of success.

Operative Intrarectal Exposure. Many surgeons, especially non-proctologists, routinely dilate the sphincters to get more room. Occasionally they have an uncomfortable period of three to six months while waiting the return of continence in some one of their patients. We believe that much more adequate exposure without the possibility of such a complication can be obtained by means of sphincterotomy. However, when as much intrarectal exposure as possible is desired, the incision should be made in either the right or left latero-anterior quadrants as this gives more room than does a posterior sphincterotomy.

SURGICAL PROCEDURE

Incision of the muscle is easily done with the patient in any of the usual rectal operating positions—Simms, prone or litho-

tomy. The tip of the left index finger is pressed into the intermuscular septum or above the pecten band if present and the subcutaneous external sphincter is palpated between it and the thumb. Then, while both buttocks are held apart and the skin of the posterior commissure is put on tension by an assistant, incision is made down the side of the finger, through the skin, through the "bar" of subcutaneous external sphincter, through the pecten band and back into Minor's triangular space lying between the two tangential legs of the superficial external sphincter. Some operators, however, like to insert a Kelly forceps between this portion of muscle and the other sphincter bundles and incise down onto the forceps.

Experience soon reveals when the internal sphincter has been reached and when all of the pecten band has been separated. Complete and adequate incision is had when the base of the wound is composed of soft, elastic muscle. No horizontal strands of fibrous tissue should be left. Finally the skin edges on each side are cut well back, (Fig. 3) and bleeding is carefully controlled. There is usually at least one active bleeder and experience has caused us to emphasize its *transfixation* as an ordinary tie has a tendency to slip off of the fibromuscular tissues. All raw areas are covered by gauze impregnated with sulfathiazol ointment and pressure is applied by means of adhesive (not a T-binder). The dressing is changed in twenty-four hours.

The anesthesia varies with the condition encountered. Most fistulas and most abscesses call for a spinal while hemorrhoids, crypts, fissures and some strictures can be done, if desired, under regional novocain infiltration accompanied by oil anesthesia in the sphincters and out in the buttocks. The latter allows the passage of a postoperative stool sooner and with less discomfort. Incidentally, we find the Angelo[32] method of obtaining an early postoperative stool without aid of oil or enema to be quite

successful. He advocated the administration of sulfathaladine pre- and postoperatively with encouragement of a spontaneous evacuation on the second or third day. The ensuing stool is soft and mushy but gives a decided signal withal.

Results are uniformly satisfactory. Healing takes place in from two weeks to two months depending on the patient, the pathologic condition and the width and depth of the incision. Often after a few months not even a scar can be seen as the posterior commissure is the site of embryonic fusion of two skin surfaces and re-epithelialization or refusion seems to take place without scar formation just as well in adult life as in embryonic life. Sometimes the whole anus becomes quite unelastic or "splinted" for three or four months but softening and relaxation eventually takes place. We have had only two patients who expelled gas involuntarily for a time. The end result in even these two patients was as satisfactory as in all the others. One clinic patient had to be given large doses of cevitamic acid before the sphincters would heal together again. Care must be exercised in the use of mineral oil as more than $\frac{1}{2}$ ounce daily may leak or produce involuntary stools before the sphincters have re-apposed. Several stools following posterior sphincterotomy for fissure in ano are not unusual due to the release of spasm, pain and fear. On the whole, we personally are so enthusiastic about this procedure that we would be at a loss to know what to do adequately without it in many situations in which we now use it as a routine measure.

CONTRAINDICATIONS

There are two conditions in which posterior sphincterotomy is contraindicated; one is functional in character and the other, anatomic. The first one is the ataxic sphincter. It is seen only rarely but not so rarely but what one of our colleagues believes that he did a posterior sphincterotomy in the presence of such a patulous
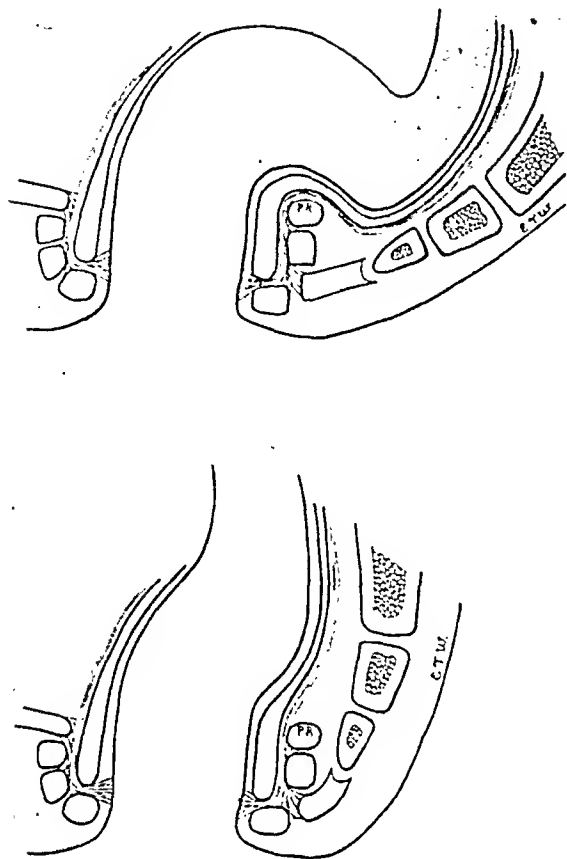
FIG. 4. Illustrating two extreme examples of a recto-anal angle. The top plate shows the constipative type. There is an acute angle at the recto-anal junction caused by a markedly concave sacrum carrying the rectal ampulla well backward while a tense puborectalis portion of the levator ani muscle pulls the anus well forward. (PR. in the illustration indicates the puborectalis, a sling-like portion of the levator ani muscle). Expulsive forces have a tendency to exert their action behind the anus rather than down through the anus. Consequently, such an acute angle places an added difficulty on the progress of a hard stool. The lower plate shows the prolapsive type. There is no angle due to a more or less vertical sacrum and a lax puborectalis sling. Prolapse of the mucous membrane or even procidentia is a frequent complication.

anus, with some incontinence as a result for an unreasonably long time. Therefore, whenever the anal musculature fails to close quickly after digital examination or whenever it is found on digital examination that the usual tonic pressure of the sphincters on the finger is lacking, the test for an ataxic sphincter should be made. This is done by forcibly inserting three fingers into the anus and deliberately separating them thereby dilating the sphincter without aid of anesthesia. If the patient accepts this insult without complaint nor attempts the escape of the fingers, then the latter are quickly removed and the anus will remain widely open especially if a little traction is made on the buttocks. The popular conception concerning this abnormal functioning of the anus puts it into the class of syphilis, but we have exhaustively examined several such cases and agree with other authors that no known etiology has as yet been found which adequately explains the findings.

The second condition is anatomic. It is the absence of an anorectal angle. (Fig. 4.) Normally the puborectalis portion of the levator ani muscle forms a sling around and behind the upper anus and lower rectum pulling them forward and thereby producing a definite angle posteriorly. The examining finger at first passes up the anus in a direction pointing toward the navel but, whenever there is a definite anorectal angle, the finger subsequently must be bent posteriorly and point toward the concavity of the sacrum due to the changed direction of the lower rectum. In extreme cases the angle is markedly acute and a definite pouch exists behind the anus anteriorly to the coccyx and sacrum. However, occasionally the other extreme is encountered wherein there is no angle and the finger continues to go straight up. (Fig. 4.) It is in such cases that prolapse and procidentia are prone to occur as this anorectal angle or shelf has a markedly rstraining influence on any tendency for the mucous membrane or rectal wall to intussuscept through the anus. In the absence of this restraining influence, nature has difficulty in preventing extrusion of the rectal wall whenever she attempts to expel rectal contents. A posterior spincterotomy would only add to nature's difficulties.

SUMMARY

The difference between posteriorsphincterotomy and pectenotomy is described,

together the accompanying anatomy and pathologic conditions, respectively.

Indications for and contraindications against posterior sphincterotomy are discussed in detail.

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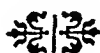
CLINICAL STUDIES OF LIVER FUNCTION

THE HEPATORENAL SYNDROME

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IN a previous study the effect of anesthetics and a variety of elective surgical procedures on liver function in eighty-four apparently healthy patients was investigated.[28] Spinal anesthesia, in the absence of a fall in blood pressure, and local anesthesia (0.5 per cent novocain) together with amytal and morphine produced no detectable alteration in the functional state of the liver following surgery. Avertin given as a basal analgesic in combination with novocain infiltration reduced liver function by 5 to 30 per cent. Open drop ether anesthesia produced transient subnormal hepatic function in all patients studied.

The present study was undertaken with the view of evaluating certain pre- and postoperative therapeutic measures directed at improving the functional state of the liver in critically ill, poor-risk, surgical patients. Because of the high incidence of associated renal and hepatic insufficiency (hepatorenal syndrome) following operation, an evaluation of the rôle of the liver in postoperative morbidity and mortality must of necessity include serial studies of both renal and hepatic function.

LIVER DEATH AND THE HEPATORENAL SYNDROME

A comprehensive review of the growing literature on the subject "liver death" and the "hepatorenal syndrome" is beyond the scope of this paper, particularly in view of the exhaustive review by Wilensky[35] and Boyce's excellent monograph on the rôle of the liver in surgery.[5] The present study, begun in 1940 and interrupted by World War ii, confirms in somewhat greater detail and extends in some instances certain of the latter author's clinical observations.

An intimate relationship between the liver and kidneys has been recognized for nearly a century; however, Heyd's description in 1924 of liver death following operations on the biliary tract appears to be the first clearcut account to justify the concept of "liver-kidney syndrome."[11] In a later analysis of mortality following operations on the biliary tract Heyd[12,13] classified his clinical material into three groups:

Type 1. In this group the patients are considered good operative risks, are not jaundiced and are subjected to simple cholecystectomy. Almost immediately from the time of operation there is a continuously ascending temperature, a rapidly developing lethargy, followed by stupor and coma with death terminating the picture in from eighteen to thirty-six hours.

Type 2. These patients are operated upon for relief of obstructive jaundice. Convalescence is normal for four or five days when, in spite of diminishing jaundice, the patient becomes delirious, then stuporous and finally comatose. Death occurs not unlike the cholemic death that occurs in unrelieved obstructive jaundice.

Type 3. These patients are not jaundiced, have what is considered normal kidney function and are considered good risks. Forty-eight hours after operation the pulse rate becomes elevated and the patient quite rapidly presents a picture of shock followed by decreased urinary output, then anuria with uremia, fever, coma and finally death.

A more detailed classification of the hepatorenal syndrome under essentially the same general headings as Heyd's has been suggested by Boyce.[5]

Boyce and others have pointed out that postoperative deaths of the above types are

in no way restricted to patients operated upon for biliary tract disease. A number of reports may be found in the literature in which the hepatorenal syndrome occurred after liver trauma[14,15,17,30] following ligation of a hepatic artery,[31] after hysterectomy,[8] after colostomy,[8] after resection of the cecum,[5] following intestinal obstruction,[35] following severe burns[3,5,7,36] and even after interval appendectomy.[5]

While the terms "liver death" and "hepatorenal syndrome" have been accepted by the majority as clinical entities, the exact nature of the breakdown of liver and kidney function remains obscure. Death follows removal of the kidneys from experimental animals due to accumulation of toxic nitrogenous excretory products. But, as yet, no investigator has been able to demonstrate the cause of death in animals with livers removed. Postmortem studies of liver and kidneys from patients whose fatal clinical course conforms with the classical picture of hepatorenal syndrome frequently reveal lesions, degenerative in character which seem inadequate to account for the fatal outcome. Touroff[32] warns against the clinical diagnosis of "liver death" in patients which develop unexplained coma and rapidly mounting fever after biliary surgery unless adequate autopsy excludes other causes. In reviewing 1,360 cholecystectomies, Touroff found only four cases which, following operation, developed without apparent cause the classical syndrome of rapidly rising temperature and shock with early fatal termination. A clinical diagnosis of "liver shock" seemed justified in all four; however, autopsy revealed an overwhelming infection to be the cause of death in all instances. Two had subphrenic abscess, one a gangrenous bronchitis with bilateral pneumonia and the other a severe hemorrhagic pneumonia.

Boyce[5] is of the opinion that hyperpyrexic liver death and liver-kidney syndrome are a single pathologic entity, the difference in the clinical course being determined by the magnitude of hepatic distintegration. Failure of hepatic detoxica-tion throws a strain on the kidneys which are in turn overcome by toxic products ordinarily rendered innocuous by a normal liver. According to this author, hepatic changes are always followed by renal lesions if the patient lives long enough.

Helwig and Orr[16] have studied in detail the blood chemistry in patients with severe traumatic liver injuries and presenting the clinical picture of the hepatorenal syndrome. Their interpretation of the causative basis for kidney failure secondary to autolysis of liver tissue differs from Boyce's only in that they ascribe the necrotic renal changes to a specific toxin of liver origin. Boyce's experimental production of degenerative changes in the liver and kidneys of dogs by the injection of saline extracts of liver from a patient who died a liver death thirty-six hours after cholecystectomy would seem in favor of a specific toxin. Control experiments in which extracts of normal liver were injected were without effect on liver or kidneys.[5]

PRESENT STUDY

In the hepatorenal syndrome two organs are involved—the liver and the kidneys. In reviewing the literature on this subject we have been impressed by the paucity of data pertaining to tests of both hepatic and renal function *before* operation or the onset of untoward symptoms. Conclusions are based in numerous instances on autopsy material, substantiated in many cases by determinations of icteric index and blood NPN during the fatal illness. The functional states of the two organs concerned *prior* to the onset of decompensation was unknown. Many writers infer normal renal reserve if the blood NPN is within normal range, a conclusion certainly unwarranted. Only after complete breakdown of kidney reserve does the NPN become elevated. Before the nature of the hepatic and renal insufficiency can be evaluated one must know something of the functional state of the respective organs *before* the onset of clinical symptoms. These conditions have

been met in the patients we have studied. Serial function tests were made before and after the onset of the hepatorenal picture.

TESTS OF HEPATIC AND RENAL FUNCTION

Liver Function.   Five tests which measured different hepatic functions were used in the present study. These were: the hippuric acid test, the bromsulfalein dye elimination test, prothrombin estimation, and in patients with T-tube drainage of the common duct, determinations of bile salt concentration in the hepatic bile. Quantitative estimations of blood bilirubin were also made in many instances.

The hippuric acid test[21] is concerned with two hepatic functions: synthesis (glycine) and conjugation (glycine coupled with administered benzoic acid forms hippuric acid). To be valid, the hippuric acid test can be used only in patients with adequate kidney function. This limits its usefulness in studying the hepatorenal syndrome. In the presence of impaired renal excretion a subnormal hippuric acid test could be the result of both hepatic and renal insufficiency, or the latter alone. The majority of our patients were too ill to tolerate the oral test. In many continuous gastric lavage was being used. In such instances the intravenous modification of the hippuric acid test was used.[24]

The bromsulfalein (BSP) dye removal test measures the ability of the reticulo-endothelial system, including the Kupfer cells in the liver, to remove injected dye from the circulation. That portion of the dye removed within the liver is transferred to the hepatic cells and is excreted in the bile. Normally a goodly portion is excreted by the kidneys. In patients with high grade *renal* retention or in the presence of a failing circulation, the results of the BSP test cannot be attributed wholly to the functional state of hepatic cells. Tests were made using the fractional modification recommended by Deutsch.[9]

Prothrombin, an enzyme or pro-enzyme essential for the normal clotting of blood,

is apparently manufactured only within the hepatic parenchyma. Liver insufficiency is accompanied by decreased prothrombin production and an alteration in the clotting time of blood or plasma.[6,23,29,34] Allen's serial dilution modification of Quick's one-stage technic was used.[1]

Determination of bile salt concentration in the hepatic bile of patients with T-tube drainage offers an extremely sensitive index of functional activity in the hepatic cells. Bile salts are manufactured from protein in the liver exclusively. Minor degrees of liver injury not detectable by the usual function tests are followed by decreased synthesis of bile salts. In states of severe liver damage their synthesis may cease entirely.[4,10,19,20,26] Bile salt determinations were made using the method of Rheinhold and Wilson[27] adapted to the photolemeter.

Blood bilirubin was determined by Hoffman's photolometric modification of the van den Bergh method.[18]

Kidney Function.   Two criteria for evaluating the functional state of the kidneys were used. Serial determinations of urea clearance[33] and blood determinations of non-protein and urea nitrogen were carried out at frequent intervals. In certain patients, quantitative determinations were made on twenty-four-hour urine collections for total, non-protein and urea nitrogen.

CASE REPORTS

For convenience in presentation the case reports are arranged in the following groups. The majority do not fit unequivocally into their respective group: (1) Early hepatic insufficiency followed by renal failure. (2) renal insufficiency with hepatic insufficiency and (3) associated hepatic and renal insufficiency.

1. *Early Hepatic Insufficiency Followed by Renal Failure.*   CASE I.   L. R., a white female, age sixty-two, was admitted on the medical service. For ten months there had been symptoms of anorexia, upper right quadrant sore-

ness, bloating, generalized weakness and a weight loss of 30 pounds. There was selective dyspepsia for greasy foods and moderate shortness of breath on exertion.

Physically the patient had a sallow, chronically ill appearance. The blood pressure was 120/80, pulse rate 96 and regular. Except for the abdominal findings and obvious weight loss the physical examination revealed nothing unusual. The abdomen was tympanitic and a soft elongated, tender mass was felt to the right of the umbilicus.

Laboratory data were as follows: On admission the hemoglobin was 75 per cent, red blood count 3,770,000, white count 14,500 with normal differential. Urinalysis revealed no unusual findings. Gastric analysis showed free HCl 39, combined acidity 43.5, total acidity 82.5. The icteric index was 10. Agglutination tests for undulant fever and typhoid were negative.

The gallbladder could not be visualized after dye. X-ray of the stomach and gastrointestinal tract revealed no abnormalities except an atonic colon. A clinical diagnosis of cholangitis was made. For thirty days the patient was treated medically. During this time the temperature spiked daily to from 100° to 102°F., frequently with an associated chilly sensation and on one occasion there was a frank chill. The upper abdominal tenderness persisted but there was no colic or severe pain. During this period of observation the icteric index fluctuated between 10 and 24.

Gallbladder drainage was advised. The patient was considered a poor surgical risk but the prognosis was considered equally poor without surgical intervention.

Further laboratory studies revealed: hippuric acid test 78 per cent, prothrombin 33 per cent, fasting blood sugar 111 mg. per cent, urea clearance 68 per cent, urine urobilinogen positive in dilution of 1:80. Although the septic temperatue continued, clinical improvement during the next eight days was observed on a regimen of high carbohydrate, low fat diet augmented by dextrose in fruit juice, bile salts (bilron), liver extract orally, vitamin B concentrate, magnesium sulfate and 1,000 cc. of 10 per cent glucose with 6.6 mg. thiamine chloride intravenously daily.

Vitamin K tablets daily for six days raised the prothrombin value from 33 to 62 per cent. (Table 1.) The preoperative hippuric acid test

TABLE I

CASE I, L. R.

Days Pre- and Postoperative	15	4	1	0	1	2	3	4	5	6	7	8	9	10	11	12	13	14	15	16	17	18	19	20
Temperature	101⁵	102²	102⁶	97⁰	98⁴	100⁰	99⁶	98⁰	98⁰	98⁰	98⁰	97⁸	98³	98³	98⁰	98³	98⁴	98⁰	98²	98⁴	98⁶	98⁶	99⁰	99³
NPN. mg. %	39	32	30		35		33					46									110			106
Hippuric acid %	78		82				70																	
Urea clearance %	68	45	70	62	54		54															22		
Prothrombin %	33	10	58				22															22		
Icteric index, units																								
Fluid intake, 24 hr./cc.				3000	1800	3000	2500	3200	4000	3300	2400	1200	2400	3500	2000	1800	2500	1500	1000	1700	2000	3000	3000	2000
Urine output, 24 hr./cc.			300	400	300	810	1200	520	570	500	600	300	780	160	380	620	370	340	340	440	710	660	550	465
Parental glucose, Gm.			100	200	100	200	150	150	100	100										100	200	250	250	150
Vitamin K		Oral 6 days		2	1	2																		
Decholin sod. 20% 10 cc.				1												1	2					1		

was 82 per cent. Both of these tests indicated only fair response of the liver to treatment. Kidney function was normal, the blood NPN was 30 mg. per cent, urea clearance 70 per cent. A 600 cc. transfusion of citrated whole blood was given the day before operation.

Cholecystotomy with drainage and an abdominal exploration was done under novocain spinal anesthesia. The liver was enlarged and mottled with purplish and yellow markings. The gallbladder was distended with approximately 16 ounces of green, watery fluid containing white sediment. This was aspirated. There were no stones. The common duct was thickened and approximately 1 inch in diameter. Palpation of the common duct and pancreas revealed nothing abnormal. One of several enlarged lymph nodes along the common duct was excised for microscopic examination. This proved to be inflammatory. Exploration of the remaining abdominal and pelvic viscera was negative.

The operative procedure was well tolerated with blood pressure maintained throughout. The postoperative course was uneventful for the first week. A blood transfusion (600 cc.) was given on the day after operation. Infusions of glucose were given daily for six days with 10 cc. of decholin sodium 20 per cent on the day of operation and on the first, second and fourth postoperative days. Decholin therapy was used to flush the infected biliary passages and to promote diuresis. (Table 1.) After the fourth postoperative day the temperature returned to normal where it remained for fourteen days.

Two hypodermic injections of vitamin K were given on the day of operation; a prothrombin value of 54 per cent was obtained on the first and again on the third postoperative days. The blood NPN was 33 mg. per cent. The hippuric acid response was reduced to 70 per cent and the icteric index was 22 on the third day after operation, indicative of adequate renal excretion but diminished liver function.

By the sixth postoperative day adequate nourishment and fluids were being taken orally, the parenteral glucose-decholin therapy was discontinued and bile salts (bilron) were given orally. Thereafter the patient gradually began to lose ground. By the end of the second week there was no desire for food. The cholecystotomy tube sloughed on the tenth day.

Clear bile continued to drain through the gallbladder fistula which had formed. Intravenous glucose and decholin was started again on the sixteenth postoperative day, but despite an accelerated urinary output (Table 1) on the seventeenth and eighteenth days, the blood NPN continued to rise. A NPN of 110 mg. per cent was obtained on the seventeenth postoperative day (blood urea nitrogen 94 mg. per cent). The patient had become drowsy and when aroused appeared utterly exhausted.

The uremic coma deepened. On the nineteenth day the skin and sclera were definitely icteric. The pulse remained between 80 and 100 per minute and, although the circulation was sluggish, there was no evidence of cyanosis. Clear bile continued to drain from the fistula. The urine output became scanty (2 ounces in twelve hours) and contained red blood cells. Later an emesis of dark coffee grounds material occurred. The prothrombin was 22 per cent, the icteric index 22. The patient expired quietly in uremic coma on the twenty-first day after operation with complete suppression of urine.

At autopsy there was no free fluid in the abdomen. The liver appeared swollen, was firm and the surface was scarred with a number of irregular gray areas 1 to 3 cm. in diameter. In the dome of the left lobe was a larger irregular, dense area measuring 3 by 5 cm. The cut surface revealed liver parenchyma infiltrated with irregular strands of rubbery, gray tissue which would not scrape off. Microscopic examination showed these areas to be organized fibrous connective tissue. Sections from the liver showed atrophy of the hepatic cords in some areas while the cells of other areas appeared large and vesicular. There was moderate periportal lymphocytic infiltration and an increase in periportal connective tissue.

The pancreas, which felt cystic before removal, proved to have most of the parenchyma replaced by flabby connective tissue. Microscopically the pancreas appeared densely fibrous. Numerous islets of Langerhans remained intact.

The kidneys were average in size and flabby and the capsules stripped fairly easily. The cortex appeared beefy red, the calices and pelves normal. Microscopically the glomeruli stood out clearly and were engorged with red blood cells. There was cloudy swelling of the tubular epithelium without evidence of de-

generation. The tubules contained many red blood cells. Anatomie diagnosis: (1) biliary cirrhosis, acute hepatic degeneration, (2) pancreatic atrophy and fibrosis, (3) mesenteric lymphadenitis and (4) hemorrhagic nephritis.

Comment. This case is our best example of the hepatorenal syndrome as described by others. At no time either during or after operation was there a period of hypotension which would introduce the factor of anoxia. As long as parenteral therapy was continued the postoperative course seemed satisfactory. When this was withdrawn the clinical course headed progressively downhill. This may or may not have been coincidence. The 8 per cent drop in prothrombin level and a normal NPN immediately after operation indicates some depression of hepatic activity and no appreciable renal impairment. By the third day jaundice was apparent and the icteric index had increased to 22 in the presence of a normal blood NPN.

In view of the pancreatic fibrosis the possibility of external pancreatic secretion deficiency as a contributing factor to liver dysfunction is a point for speculation. The liver sections showed in addition to acute degenerative changes considerable fatty degeneration. It is conceivable in this particular patient that prolonged impairment of hepatic detoxication threw an unburdenable load on the kidneys. It is noteworthy that this patient showed none of the circulatory or central nervous system manifestations which characterized the postoperative course of the majority of patients in this series.

CASE II. M. H., a female, age fifty, entered on the medical service complaining of gallbladder trouble and yellow jaundice. The present illness dated back four months during which interval there had been recurrent attacks of upper right quadrant, colicky pain, anorexia, epigastric fullness relieved by vomiting, jaundice and severe itching. There was a history of jaundice of nearly a year's duration in 1931 with symptoms comparable to those of the present attack. This cleared up and for three years the health was relatively good. In 1934 the patient had another series of attacks of knife-like, upper abdominal pain and jaundice. A course of "fruitola" was taken and after a night of cramping pains several large gallstones were recovered from the stool. These were in the patient's possession and were true gallstones of the calcium-pigment variety. The largest measured 15 mm. in diameter. Following the passage of the stones the symptoms and jaundice abated. Only minor gastrointestinal distress was experienced during the next six years.

During the four months preceding hospitalization there had been a weight loss of 40 pounds and the patient had become more irritable. The remainder of the history was irrelevant.

Physical examination revealed a poorly nourished, moderately jaundiced white female who appeared ill. The temperature was 99°F., pulse 90 and regular, blood pressure 115/65 mm. The abdomen was soft with marked tenderness and a questionable mass in the upper right quadrant. The remaining findings were not remarkable. A flat plate x-ray of the gallbladder area revealed nothing.

Laboratory data were as follows: Hemoglobin 70 per cent, red blood count 3,080,000, white count 16,150 with normal differential Routine urinalysis was negative A PSP test for kidney function gave 55 and 15 per cent for the first and second hours. Mosenthal test: specific gravity 1.005 to 1.017. The icteric index was 37.

The patient was observed and treated for two weeks with a high carbohydrate-low fat diet, daily infusions of 100 Gm. glucose, hot packs to upper abdomen and nitroglycerin sublingually for pain. Two 500 cc. transfusions of citrated blood were given. During this period the temperature fluctuated between normal and 100°F. and the icteric index declined from 37 to 21. An intravenous hippuric acid test was 46 per cent, the prothrombin time 116 per cent. The patient was transferred to the surgical service and was treated for an additional two weeks. Intravenous glucose was discontinued for four days and then resumed, 100 Gm. daily for ten days. The following additional therapeutic measures were instituted daily: glycine in fruit juice, liver extract orally, bile salts and hematinic plastules. An intramuscular injection of concentrated liver

extract was given daily for seven days. During this time normal prothrombin values prevailed; however, the hippuric acid test values declined progressively. (Table II.) The blood NPN was 30 mg. per cent. The patient had remained free of abdominal distress, except bloating. A faint icterus persisted, accompanied by itching of

ing 250 Gm. glucose and 1 ampule decholin sodium (10 cc. 20 per cent). Throughout the remaining postoperative period 200 Gm. glucose and 1 ampule decholin sodium were given intravenously daily.

During the second twenty-four-hour period the output was reduced to 120 cc. from an

TABLE II
CASE II. M. H.

Days Pre- and Postoperative	31	28	26	23	19	15	13	10	4	0	1	2	3	4	5
Temperature (max.)	99^0	100^0	98^0	100^0	99^0	100^2	99^0	99^0	98^6	98^6	99^6	99^0	100^6	100^2	101
NPN, mg. %	..	..	..	..	..	...	..	30	..			51		60	
Hippuric acid test %	..	..	..	..	46	...	20	18	22	Operation					
Prothrombin %	..	..	..	..	..	116	..	102	..						
Icteric Index	37	30	23	23	33	20	..	...	21			86		120	
24 hr. fluid intake cc	..	..	..	..						3000	2000	3000	2000	2000	
24 hr. urine output cc	..	..	..	..						240	120	355	1350	1500	550
Bile drainage cc	..	..	..	..						400	300	480	320	120	
Decholin, 20%, 10 cc	..	..	..	..						1	1	1	1	1	
Parenteral glucose, Gm	..	..	..	..						300	200	150	150	150	

the hands and feet. A purpuric area approximately 2 by 3 inches appeared spontaneously on the left forearm. Bleeding, clotting and prothrombin times were normal. The platelet count was 174,000. The preoperative hippuric acid test remained critically low (22 per cent) indicating irreparable liver damage.

Laparotomy and gallbladder drainage under procaine spinal anesthesia was done thirty-one days after admission. The gallbladder was distended with white bile which was aspirated. Two large pigmented stones and cholesterol gravel were removed. Approximately one liter of cloudy ascitic fluid was aspirated from the abdominal cavity. The liver was subnormal in size, grey-blue in color, very hard and of hobnail appearance. The common duct was involved in adhesions and was left undisturbed because of the patient's poor condition. Catheter drainage of the gallbladder was provided. Fifteen minutes after the spinal anesthetic the blood pressure dropped to 86/54. Adrenalin by hypo, oxygen by mask and intravenous glucose solution relieved the hypotension promptly. Immediately after operation a transfusion of 500 cc. citrated whole blood was given.

During the twenty-four-hour period following operation the general condition seemed satisfactory; however, the urinary output was but 240 cc. from an intake of 3,500 cc. contain-

intake of 2,000 cc. Three hundred cc. of bile drainage was collected during this period. Nasal oxygen which had been given twenty minutes of each hour since operation was discontinued. Forty-eight hours after operation the icteric index had become elevated to 86 and the NPN was 51, indicating impairment of both liver and kidney function. The patient was very restless and complained of severe itching. Nausea requiring gastric lavage every six to eight hours had developed. On the second postoperative day (third twenty-four-hour period) the patient seemed more alert and was less nauseated, but the itching continued and the jaundice was more apparent. The urinary output increased to 355 cc. On the third day 1,350 cc. of urine was excreted; however, the course continued downhill and the NPN continued to rise. Despite free bile drainage from the cholecystotomy tube and the increased urinary output which continued on the fourth day (Table II) the icteric index reached 120 and the NPN 60 mg. per cent. The patient had become very restless and was confused at times. Continuous gastric lavage was necessary. Persistent cyanosis despite oxygen therapy developed. The pulse and blood pressure could not be obtained before respiration ceased.

Comment. This case is a classical example of hepatorenal syndrome. Before

operation we were dealing with advanced hepatic insufficiency, at least in the function of detoxication, which could not be improved by specific therapeutic measures. Operation was followed by evidence of increased liver damage and renal decompensation with urinary suppression and azotemia. The degree of renal insufficiency was of such magnitude that, despite an induced diuresis on the third and fourth postoperative days, nitrogenous retention was progressive. The terminal picture was one of deepening icterus, uremia and circulatory collapse.

Case III. B. T., a female, age twenty-seven, was referred to the surgical service for splenectomy. The patient had contracted malaria at the age of six years which was apparently cured with quinine. The next thirteen years were uneventful. At the age of nineteen she suffered a severe case of Vincent's angina which resulted in the loss of most of the teeth and a swelling in the neck that required incision and drainage. Two years later the patient began to develop intermittent attacks of pain under the left costal margin. These attacks which occurred once or twice a year or oftener, were always followed by varying degrees of jaundice. She was hospitalized on two occasions over a three-year period with diagnoses of splenic anemia on one admission and chronic malaria on the other. Intramuscular atabrine was followed by improvement; however, during the next four years her health was not good. A change in climate was advised and she moved from Arkansas to Michigan a year before the present admission. For a few months she felt well and gained weight. Six months before admission abdominal swelling thought to be due to fluid developed. Several months later the patient was considerably weakened by loss of blood which was expelled by emesis and in the stool. Seven blood transfusions were given. Biopsy of liver and spleen was done at the University Hospital at Ann Arbor, Michigan, with a diagnosis of Banti's disease. Splenectomy was recommended. Paracentesis had been done on three occasions, the largest amount of fluid being 3 pints.

Physical examination revealed a restless, fairly well developed, emaciated, deeply jaundiced white female. Most of the teeth were missing. The essential findings were decreased area of liver dullness, an enlarged spleen which filled most of the upper left quadrant and a moderate amount of fluid within the abdominal cavity.

Laboratory data were as follows: Hemoglobin 75 per cent, red count 3,790,000, white count 4,250 with a relative lymphocystosis. Over a five-day period daily blood smears for malaria were negative. The platlet count was reduced to 55,040. Bleeding and coagulation time were normal. Fasting blood sugar was within normal limits. The hippuric acid test was 32 per cent, icteric index 120, serum protein 7.5 Gm., blood NPN 30 mg. per cent, and urea clearance decreased to 62 per cent. Routine urinalysis was negative except for one cast.

During five days of preparation for surgery the patient experienced two mild chills without fever. Preoperative treatment included intravenous glucose with thiamine and ascorbic acid, oral bile salts, liver extract and glycine.

At operation under novocain spinal anesthesia the spleen filled the upper left quadrant. The liver was small and uniformly hob-nailed. Splenectomy was done. There was a transient fall in blood pressure to shock levels that responded promptly to subcutaneously administered adrenalin. Oxygen-carbon dioxide was given by mask throughout the operation.

Immediately after operation a 500 cc. citrated blood transfusion and 1,000 cc. 10 per cent glucose were given. This was followed by 2,000 cc. 5 per cent glucose in water by hypodermoclysis. The twenty-four-hour urinary output from this intake was 200 cc. Blood pressure was maintained for the first eighteen hours and then declined to 64 ?. The pulse rate was 140 per minute. The patient was stuporous. Following 500 cc. plasma-glucose and 500 cc. 20 per cent glucose with 1 ampule of decholin the blood pressure increased to 110/60 with a pulse rate of 112. Three hours later the patient was semicomatose with a blood pressure of 60/25 and an apex rate of 160. Oxygen by nasal catheter was begun and 500 cc. of citrated blood by vein elevated the blood pressure to 90/30. The pulse rate was 140. The patient was comatose. The eyes were open but there was no response. During the second twenty-four hour period the urinary output was 220 cc.; however, only 40 cc. were obtained during the last ten hours of the period.

Forty-eight hours after operation the patient was in coma and was deeply jaundiced. The blood pressure was 77/?, apex rate 160. Five hundred cc. of plasma and 500 cc. 20 per cent glucose with calcium gluconate raised the blood pressure to 110/50. Over a six-hour period 90 cc. of urine was excreted. Within two hours after the plasma infusion the blood pressure declined to 90/40 with a pulse rate of 154. Spells of muscular twitching which developed into clonic convulsions lasting two to three minutes followed. The temperature, which had fluctuated between 100° and 101°F. during the preceding twenty-four hours, climbed to 105°F. and the patient expired quietly in circulatory collapse.

Comment. This patient, like Case II, had advanced portal cirrhosis with significant hepatic insufficiency as measured by the hippuric acid test. Portal cirrhosis is characterized pathologically by patchy involvement of hepatic parenchyma and physiologically by relatively well maintained hepatic function until the disease is well advanced. Areas of connective tissue replacement alternate with areas of normal or even hyperplastic hepatic cells. Because of the large factor of safety possessed by the liver only a fraction of normal parenchyma can maintain hepatic function. On the other hand, diffuse hepatic lesions which compromise the entire parenchyma give evidence of diminished function early.

Both Cases II and III gave laboratory evidence of depleted hepatic reserve before operation. It is not unlikely that the kidneys in these patients were subjected to toxic products which would have been rendered inocuous by a normal liver. The profound circulatory instability exhibited by Case III was an outstanding feature of the postoperative course.

2. *Renal Insufficiency with Hepatic Insufficiency.* CASE IV. F. G., a female, age sixty-four, was admitted to the hospital five months after permanent drainage of the gallbladder elsewhere for a partial common duct obstruction. The drainage tube had been clamped off most of the time, but on occasions there were spells of upper right quadrant distress with nausea. These were always relieved by unclamping the tube to the gallbladder which was followed by drainage of several ounces of bile. Following the first operation at which time the patient was deeply jaundiced much of her former vitality had been regained. Of late there had developed anorexia, loss of strength and foamy, clay-colored stools off and on. The intervals between spells of upper abdominal distress had diminished progressively to every few days.

Except for attacks of gallbladder colic for two years prior to the onset of painful, complete obstruction of the common duct which led to the above operative procedure, the past history was irrelevant. There were no symptoms referable to the cardiovascular and renal systems.

Essential physical findings revealed an elderly white female who appeared somewhat malnourished and whose skin had a leathery, brownish-yellow tinge. The blood pressure was 122/70, the pulse 64 and regular. Heart and lungs were negative. A rubber drainage tube protruded through an indurated, healed upper right quadrant scar.

Laboratory findings on admission were as follows: Hemoglobin 89 per cent, red blood count 4,300,000, white blood count 8,750. Bleeding time two minutes, coagulation time four minutes, prothrombin time 89 per cent. Blood NPN 30 mg per cent, urea N 12 mg. per cent, serum protein 5.7 Gm. The urea clearance test showed impaired kidney function (55 per cent). Icteric index was elevated to 24, fasting blood sugar was 92 mg. per cent and blood amylase at the lower limits of normal (66 units, Somogyi). A subnormal hippuric acid test of 72 per cent on admission was raised to 89 per cent following six days of preparation for operation. With the bromsulfalein test, 40 per cent of the dye was retained after thirty minutes.

Following lipiodol injection of the biliary tract through the cholecystostomy tube which produced typical gallstone colic pain a transient elevation of temperature (102°F.) and an elevated blood amylase was observed (242 units). After five days of therapy, designed to build up hepatic reserve and combat bile salt and pancreatic deficiency, exploration of the common duct under spinal anesthesia was done. Much débris and two faceted stones were removed from the ampulla and T-tube drain-

age of the common duct was provided. The operative procedure was tolerated well.

For the first seventy-two hours after operation the urinary output was diminished. (Table III.) On the first postoperative day the blood pressure declined to 82/54. Following hepatic damage contributed to the clinical picture. On the morning of the sixth postoperative day the NPN dropped to 84 mg. per cent and during this and the following day the twenty-four-hour urinary output was 2,000 cc. Clinical improvement followed and gastric

TABLE III
CASE IV. F. G.

Days Pre- and Postoperative	7	4	1	0	1	2	3	4	5	6	7	8	9	10	11	12	13
Temperature	98^0	99^0	97^8	98^0	99^0	98^0	98^4	98^4	98^4	98^4	98^4	98^6	98^0	98^0	98^8	98^4	98^4
NPN mg. %	30	...	...	....	....	88	102	116	128	84	65	....	56	50	47	42	34
Urea clearance %	55	...	...	....	....	....	12										
Hippuric acid %	72	...	89														
Prothrombin %	89	...	...	....	....	80	....	....	90								
Icteric index, units	24	...	...	....	....	16	....	....	....	....	....	....	....	....	....	....	9
BSP test, %	40	...	...	....	....	0											
Fluid intake, 24 hr./cc	..	...	...	2500	2500	3000	3000	1800	2000	3800	2750	3000	2200	1500	2000		
Urine output, 24 hr./cc	..	...	...	300	750	500	1870	920	1500	2000	2000	1580	820	820	1420	1110	800
Parentenal glucose, Gm	..	100	100	200	200	300	250	200	200	200	100	100	100	....	100		
Decholin, 20%, 10 cc	..	...	...	1	1	1	1	1	1	1	1	1	1	....	1		

intravenous fluids, circulation improved and for the next fourty-eight hours the pressure varied between 108/70 and 122/74 mm. The pulse rate never exceeded 90 per minute. It was necessary to provide continuous gastric lavage which, however, did not allay spells of singultus. Intravenous decholin sodium was started on the first postoperative day and was continued along with intravenous and subcutaneous glucose for nine days. (Table III.) On the second postoperative day there was no retention of bromsulfalein thirty minutes after injection of the dye and the prothrombin time was on 9 per cent under the preoperative value, indicating no appreciably increased impairment of hepatic activity as reflected in these two hepatic functions. A blood NPN of 88 mg. per cent and a urea N of 48 mg. per cent suggested rather marked impairment of kidney function. A progressive azotemia developed in spite of increasing urinary output and the clinical picture was that of uremia. The greatest nitrogen retention occurred on the fifth postoperative day (NPN 128 mg. per cent, urea N 80 mg. per cent). At this time the prothrombin value was 90 per cent of normal. Inasmuch as this corresponds to the preoperative prothrombin level and since no vitamin K had been administered postoperatively, it seems unlikely that any significant degree of generalized lavage was discontinued on the seventh postoperative day. Thirteen days after operation the nitrogenous constituents of the blood and icteric index were normal and the patient recovered rapidly.

Comment. This patient is one in whom, in view of the facts related to the biliary tract disturbance, one would anticipate a narrow margin of hepatic reserve. The clinical picture after surgery conforms with that frequently classified as "hepatorenal," yet two hepatic functions (prothrombin and dye removal) gave no indication of hepatic insufficiency on the second postoperative day. From blood chemistry and urinary output data the azotemia which developed would seem most satisfactorily explained on the basis of impaired kidney function due to renal disturbance. The impaired urea clearance before operation favors this view.

3. *Associated Hepatic and Renal Insufficiency.* CASE v. R. S. M., a male, age seventy-six, entered the hospital with a suprapubic catheter in the bladder. Eleven days previously the patient had experienced acute urinary retention due to benign prostatic hypertrophy which had caused some symptoms for a year.

The patient was almost deaf and a detailed history was not obtained; however, it was learned from the family that the patient's general health had always been good and there had been no serious illnesses or gastrointestinal symptoms. There was occasional dizziness and moderate dyspnea on exertion.

Physical examination revealed a well preserved, elderly white male who was alert and not acutely ill. The pulse rate was 76, respiration 18, blood pressure 120/70 mm. The teeth were in poor condition, the heart and lungs seemed normal, the abdomen was soft without masses or tenderness, the suprapubic incision was well healed. By rectal examination the prostate was of the size, shape and consistency of a baseball.

Laboratory data were as follows: Hemoglobin 80 per cent, red blood count 4.23 million, white blood count 5,050 with a normal differential; urine, specific gravity 1.020 with a trace of albumin and many pus cells. Blood NPN 42 mg. per cent, urea nitrogen 22 mg. per cent, and urea clearance 58 per cent, indicating impaired kidney function. Prothrombin was 92 per cent and there was no retention of bromsulfalein in thirty minutes, indicating no impairment of liver function.

The bladder infection was treated for one week with sulfanilamide and 500 cc. infusions of 20 per cent glucose with 10 cc. decholin sodium 20 per cent were given daily for seven days to promote diuresis. Following this, decholin tablets (twelve daily) were given for four days. The blood NPN which reached a maximum elevation of 68 mg. per cent two days after beginning the intravenous therapy declined to 39 mg. per cent ten days later.

At operation suprapubic prostatectomy was done without difficulty under novocain spinal anesthesia. Three hours after operation the blood pressure was 70/56, the pulse rate 100 and rather weak. The dressings were soaked with sanguinous drainage but there was no evidence of undue hemorrhage. Following the intravenous injection of 500 cc. 20 per cent glucose with decholin, ephedrine by hypodermic and oxygen by nasal catheter the blood pressure rose to 115/70. Five hours later the pulse was weak and thready and the blood pressure could not be obtained. A 400 cc. citrated whole blood transfusion was without effect on the low blood pressure. The pulse was somewhat irregular. Two hypodermic injections

of ephedrine (50 mg.) and two ampules of digitalis given an hour apart elevated the blood pressure to 76/42. An ampule of pitressin by hypodermic raised this to 94/64. Fourteen hours after operation the patient appeared slightly jaundiced and during the night there were several emesis, the last of which was blood tinged. The pulse remained poor in quality. Twenty-four hours after operation the patient was semicomatose, there was moderate jaundice, a blood pressure reading could not be obtained, and a gastric lavage contained coffee ground material. Continuous gastric drainage was instituted. Following 500 cc. of plasma-glucose and 500 cc. 20 per cent glucose with decholin the blood pressure rose to 150/85, the pulse became stronger and the patient looked and acted greatly improved. This state was maintained throughout the first postoperative day. The blood NPN was 70 mg. per cent. The urine output was 400 cc. for the first twenty-four hours and 1,300 for the second twenty-four-hour period. Thereafter permanent urinary suppression set in. On the second postoperative day the temperature was 99°F, the blood NPN was 108 mg. per cent, there was singultus at intervals and cyanosis was apparent in spite of continuous oxygen therapy. Fluid intake was maintained parenterally. On the evening of the second day the patient was stuporous, the icterus was more pronounced (icteric index 80), the blood pressure was 70/?, the pulse rate 130 and poor in quality.

On the third postoperative day the temperature reached 101°F, the blood NPN was 135 mg. per cent, singultus continued, the pulse was 144 and thready and the blood pressure could not be obtained except for several hours following intravenous infusions. On the morning of the fourth day the patient was in deep coma, there were moist râles throughout the lungs and the skin and sclera were deeply jaundiced. The blood pressure could not be obtained. The temperature increased to 104°F and the patent expired ninety-six hours after operation.

At autopsy the skin was deep yellow. The abdominal cavity appeared normal. The prostatic bed contained an adherent blood clot. The liver was larger than average, the borders were rounded and the surface appeared dusty yellow. The cut surface had a greasy appearance and felt granular when scraped. Microscopically the liver cords were shrunken throughout; in many areas the normal pattern

was lost with the hepatic cells appearing large and vesicular and arranged in clumps.

The kidneys were small, the surfaces scarred and the capsules adherent. Cut surface revealed narrowing of the cortex with indistinct markings. Under the microscope the glomeruli were shrunken and their epithelium showed advanced sclerotic changes. The cells of the tubules were swollen and clear; in some areas the protoplasm took almost no stain. Desquamation and other advanced degenerative changes in the tubular epithelium were scarce.

The lungs were wet but contained no areas of consolidation. The remaining findings were irrelevant.

Comment. This case illustrates a combination of acute hepatic degeneration followed promptly by renal failure in a patient with a normal NPN but no renal reserve. The liver damage accompanied by jaundice within twelve hours of operation may or may not be related to the pronounced tendency to surgical shock. The absence of fever during the first forty-eight hours after operation makes the possibility of a blood stream infection unlikely. The vasomotor instability was the most outstanding clinical feature of the postoperative course.

Decholin therapy before operation produced a diuresis but the elimination of urinary nitrogen (twenty-four-hour periods) was not increased, suggesting that any benefit from decholin in azotemia results from some mechanism other than increased urinary excretion of nitrogen bodies.

The following case is interesting both from a diagnostic standpoint and in its postoperative hepatorenal aspects:

CASE VI. F. J., a white female, age forty-nine, was admitted to the medical service and transferred to the surgical service a week later. Three weeks prior to admission the patient had an attack of what was thought to be the "flu." There was generalized muscular aching with loss of appetite but, it was believed, no fever. The patient remained in bed for two days but did not regain her former strength or appetite.

One week after the above illness the patient's family noticed that her eyes and skin were turning yellow. The patient denied any abdominal distress or attacks simulating colic. The appetite had been only fair, and for several years there had been occasional spells of gas and bloating. The bowels had been costive as a rule for a number of years, but there had been no noticeable change in bowel habit and no bloody or tarry stools. The remaining past history was irrelevant except that her father died of carcinoma of the liver.

Physical examination revealed nothing of diagnostic value. The patient was alert, intelligent and cooperative. There was an icteric tint to the skin and sclera, the lungs and heart appeared normal, the blood pressure was 165/100, the pulse 80 and regular. No abdominal masses or tenderness were made out.

A preliminary diagnosis of catarrhal jaundice was made. An intravenous hippuric acid test of 14 per cent and a prothrombin value of 76 per cent that declined to 45 per cent four days later was thought to substantiate the diagnosis of intrahepatic jaundice. However, flat plate x-ray of the gallbladder region revealed one large and two smaller positive shadows and following three days of vitamin K therapy (kayquinone) with desoxycholic acid* orally, the prothrombin time promptly rose to 90 per cent of normal and the diagnosis was changed to obstructive jaundice, probably due to a silent common duct stone. This diagnosis seemed further strengthened by the fluctuating icteric index during nine days of observation (Table IV.)

The patient responded promptly to a high carbohydrate high protein diet, bile salts and vitamin K and intravenous glucose, and before operation the prothrombin was normal and the hippuric test (intravenous) was 70 per cent. The blood NPN was 30 mg. per cent, urea clearance decreased to 45 per cent (25 per cent below the considered normal of 70 per cent), hemoglobin 77 per cent, red blood count 3.73 million, white blood count 5,550 with normal differential and the urine negative except for the presence of bile. The icteric index had increased to 150; quantitative blood bilirubin was 12.0 mg. per cent (normal 0.2 to 0.4 mg. per cent).

Operation was performed under novocain spinal anesthesia. The gallbladder and common

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* Decholin sodium and desoxycholic acid (degalol) used with vitamin K for differentiation of intra- and extrahepatic jaundice on the basis of prothrombin response has been kindly supplied by Riedel de Haen and Co., New York.

duct were only questionably distended and the contained bile was pale green and thick with mucus. The common duct was explored and found to contain one pea-sized stone. Several stones were removed from the gallbladder and tube drainage was provided for both the gall-

considering the almost complete blockage of both liver and kidneys. The blood NPN had risen to 76 mg. per cent, and the prothrombin had dropped from 100 per cent on the second postoperative day to 58 per cent. Vitamin K was given by hypodermic injection for four

TABLE IV

CASE VI. F. J.

Days Pre- and Postoperative	8	6	3	2	1	0	1	2	3	4	5	6	7	8	9	10	11	12	13
Temperature	98^1	98^4	98^4	98^4	98^4	98^4	99^2	100^2	100^2	98^1	98^0	99^0	98^4	98^2	98^2	98^0	98^5	97^6	98^4
NPN mg. %					30				72	76	88	110	110		106	114	136	136	
Urea clearance %					45														
Hippuric acid %	14		42		70														
Prothrombin %	76	45		80	90			90	65	58	80	84	90	70	52	40			
Icteric index, units	32	20	110	100	150			200			220		250				250		
Fluid intake, 24 hr./cc						3400	3750	2000	4200	3500	1500	1500	1500	2500	2500	1250	1800	1000	
Urine output, 24 hr./cc							100	80	150	40	20	65	0	25	30	45	60	40	65
Parenteral glucose, Gm						200	150	200	250	250	150	200	150	200	100	150	150	150	
Vitamin K-bile salts—oral*		4 days																	
Vitamin K-hypo										1	1	1	1						
Decholin, 20 cc. 10%							1	2	1	1									

* Kayquinone Abbott 75 mg. and Degalol (Reidel de Haen) 18 gr.

bladder and common duct. Exploration of the pancreas and gastrohepatic omentum revealed in the latter a hard nodule the size of a hazelnut which was taken for microscopic examination. The upper surface of the liver was smooth; however, palpation of the under surface revealed multiple hard nodules beneath the capsule and surrounding the bifurcation of the common bile duct. The lower abdomen was then explored and a hard, circumscribed neoplasm was found in the large bowel just distal to the splenic flexure.

During the operative procedure there was a transient fall in blood pressure from 125/84 to 80/20. Adrenalin, 0.5 cc. by hypo and 95 per cent oxygen-carbon dioxide 5 per cent by mask properly restored the pressure to 140/100; during the following thirty minutes this declined slowly to 100/78 where it was maintained.

Twelve hours later the patient was resting quietly, aroused easily and was taking fluids well. The blood pressure during the first twenty-four-hour period was maintained at 100/65, the pulse rate 120 and regular, the urinary output 100 cc. and from this time on an almost complete suppression of urine ensued. (Table IV.) There was likewise essentially no bile drainage. Hypertonic glucose and decholin were without effect on the anuria. On the fourth postoperative day it was necessary to institute continuous gastric drainage; however, the patient's general condition was amazingly good

consecutive days and a prompt response in prothrombin time to 90 per cent was observed on the seventh postoperative day. Following withdrawal of vitamin K therapy the prothrombin value declined progressively to 40 per cent.

By the eleventh postoperative day the patient was dull and exhausted with spells of nausea and singultus. The breath had a uriniferous odor and the blood NPN was elevated to 136 mg. per cent. Except during the first twenty-four hours postoperatively, the blood pressure remained above 120 mm. systolic and the temperature was essentially normal. Clinically, death on the thirteenth postoperative day appeared to be due primarily to uremia.

Postmortem examination revealed complete walling off of the gallbladder and common duct region with omental adhesions. There was no evidence of bile or infection in the abdominal cavity. The liver showed gross and microscopic evidence of early obstructive biliary cirrhosis with distended bile capillaries, increased pigmentation and varying degrees of central atrophy of liver cells. Microscopically the nodules in the liver hilus and that removed at operation from the gastrohepatic omentum showed the same metaplastic cellular structure interpreted as metastatic from the adenocarcinoma in the large bowel. The kidneys were small and scarred, the capsules stripping with difficulty. The microscopic picture was that

of fairly advanced chronic glomerulonephritis with varying degrees of hyaline degeneration of the glomeruli and atrophy of many of the collecting tubules. Pigmented débris and degenerative cellular changes were prominent throughout the tubular areas.

Comment. This case is extremely interesting in that observations were made over a thirteen-day period during which there was essentially complete obstruction to the liver and an almost complete suppression of urine. During the first half of this period the general condition appeared unbelievably good under the circumstances. It is noteworthy that during this period the obstructed liver still responded promptly to vitamin K therapy, a response not obtained in the presence of intrinsic rather than extrinsic jaundice. It seems unlikely that a rapidly failing liver would manufacture prothrombin so promptly. On the basis of this response it would seem that despite the clinic resemblance to the hepatorenal syndrome, hepatic insufficiency played little if any part in the marked urinary suppression observed during the first seven or eight days following operation. On the other hand, the degree of chronic glomerulonephritis present undoubtedly narrowed significantly the degree of renal reserve. Although there was no nitrogenous retention before surgery, the lowered urea clearance test (45 per cent) is evidence of a lowered renal factor of safety. The following explanation is offered to account for the course of events which followed surgery in this patient:

The transient fall in blood pressure during operation and the relatively low pressure for the next twelve to sixteen hours was sufficient in this particular individual, whose original blood pressure was 165/100, to produce a lethal anoxia in the remaining functional kidney parenchyma. Degenerative changes resulting from this damage, coupled with the added load of bile excretion, was sufficient to plug the remaining functional collecting tubules to the extent that the excretion of urine was reduced almost to nothing. (Table IV.)

CASE VII. The patient, H. B., a female, age sixty, was transferred from the medical service with a diagnosis of acute cholecystitis with lithiasis and obstruction of the common duct, partial. The past history was one of gas and bloating with belching and burning in the epigastrium of fifteen years' duration. Eight days before admission the patient had an attack of severe gallbladder colic requiring several hypodermic injections of morphine for relief. Following this marked jaundice developed. On admission the icteric index was 100, blood NPN 32.79 mg. per cent and the hippuric acid test 71 per cent. Surgical intervention was refused and after eleven days of medical management the patient was dismissed on a high carbohydrate diet and bile salts. On dismissal the icteric index had declined to 75.

The patient was readmitted on the surgical service two weeks later. The jaundice had become more intense, dyspepsia for all types of food had developed along with recurrent attacks of upper right quadrant pain associated with distention and vomiting.

Essential physical findings revealed an obese, white female with yellow sclera and a canary yellow skin. There were no positive chest findings, the blood pressure was 130/80, the pulse 64 and regular. The abdomen was domed and tympanitic, there was marked tenderness without rigidity in the upper right quadrant and epigastrium, and a smooth tender mass the size of an orange was palpable under the right costal margin. The temperature was 98°F.

Laboratory findings were as follows: Hemoglobin 95 per cent, red blood count 4.84 million, white blood count 11,500 with an increase in segmented forms. Mosenthal test 1.004 to 1.016, NPN 30 mg. per cent, urea clearance test 56 per cent (subnormal). The prothrombin time was 62 per cent of normal. The icteric index was 200. During three weeks the hippuric acid test had dropped from 71 to 31 per cent.

The patient was placed on a high protein, high carbohydrate diet with Karo syrup in fruit juice, bile salts (bilron) and 1,000 cc. of 10 per cent glucose with 1 Gm. of calcium gluconate, 6 mg. of thiamine chloride and 100 mg. of ascorbic acid intravenously daily. Nitroglycerin, gr. 1/150 sublingually and atropine sulfate, gr. 1/75, was given three times daily on alternate days. The response was poor due to nausea and vomiting whether food was taken or not. It was decided that gall-

bladder drainage offered the only chance for improvement.

Under local novocain infiltration anesthesia the abdomen was opened by an upper right gridiron incision. The liver appeared enlarged with borders rounded, greyish in color, and

systolic. One hundred sixty-five cc. of urine were obtained following fluid intake of 3,225 cc. containing 200 Gm. of glucose. At the end of this period the blood pressure was 105/65, the pulse rate 90, respiration 18. The general appearance seemed satisfactory and oxygen

TABLE V

CASE VII. H. B.

Days Pre- and Postoperative	6	5	4	3	2	1	0	1	2	3	4	5	6	7
Temperature	99^2	98^6	98^0	98^4	98^4	98^8	98^0	98^4	98^0	98^0	97^4	98^6	97^2	97^0
NPN mg. %	...	30	..	...	..	..	...	...	50	...	...	...	90	
Urea clearance %	...	..	56											
Hippuric acid %	...	..	..	31										
Prothrombin %	...	..	..	62	..	..	...	...	43	...	...	...	30	
Icteric index, units	200	..	..	170	..	..	...	...	200	...	...	...	200	
Bile drainage, cc.	...	..	..	...	..	..	200	200	550	625	525	850	350	
Cholates in drainage bile, mg. %	...	..	..	...	..	..	...	120	...	100	...	...	86	
Fluid intake, 24 hr./cc.	...	..	..	...	..	..	3225	3500	2500	2270	2000	1500	1500	
Urine output, 24 hr./cc.	...	..	..	...	..	..	165	90	275	270	930	1030	90	
Parenteral glucose, Gm.	...	..	..	...	..	..	150	200	150	200	100	...	100	
Decholin, 20%, 10 cc.	...	..	..	...	..	..	...	...	2	1	1			

mottled with yellow spots the size of a pin head. The gallbladder was distended to about four times average size; aspiration yielded 6 ounces of colorless fluid. Upon excising the dome of the gallbladder several ounces of dark green, mucoid material loaded with gravel was obtained. A number of stones were removed from the gallbladder and cystic duct and a rubber drainage tube was fixed in the gallbladder with purse-string suture. The common duct which was markedly distended was not disturbed. There was no evidence of metastases along the course of the common duct.

The operative procedure, which required thirty-two minutes, was well tolerated except for a persistently low blood pressure (70/40 to 90/60 mm.); 95 per cent oxygen-5 per cent carbon-dioxide was given by mask throughout the operation. At the end of the procedure clear, pigmented bile was draining from the cholecystotomy tube.

Following operation oxygen was given by mask (6 liters per minute) twenty minutes of each hour for the first twenty-four hours. Postoperative temperature, laboratory tests, fluid intake and output and volume of bile drainage are shown in Table v.

During the first twenty-four hours the blood pressure varied between 70 and 80 mm.

inhalations were discontinued. For the next twenty-four hours the patient was anuric in spite of an intake of 3,500 cc. The urinary output for the second twenty-four hour period was 90 cc. There was free bile drainage; however, the bile salt concentration for the first twenty-four hours was 380 mg. per cent (normal around 2,000 mg. per cent). Nausea and occasional emesis persisted, the jaundice appeared to be increasing, the blood pressure remained around 100 mm. systolic, the pulse rate varied between 70 and 88 and there had been no elevation of temperature.

Following hypertonic glucose and decholin (Table v) the urinary output increased to 275 and 270 cc., respectively, on the second and third postoperative days. The icteric index was 200, the blood NPN had increased to 50 mg. per cent. On the fourth and fifth postoperative days the urine output was increased to 930 and 1,030 cc. It was hoped that with improved renal elimination some clinical improvement might follow. Such was not the case. The blood pressure declined to between 70 and 80 mm. systolic and the patient appeared weakened and semistuporous. A 600 cc. citrated whole blood transfusion produced no change in the general condition or blood pressure level. For the last twenty-four hours of

life the patient was in deep coma, and during twenty hours prior to death no urine was eliminated. The blood NPN was 90 mg. per cent on the sixth postoperative day. A free drainage of highly pigmented bile continued to the end, but on the sixth postoperative day the concentration of bile salts was critically low (86 mg. per cent).

Comment. This patient presented early hepatic insufficiency with delayed renal failure. Here again we encountered a very early oliguria which was later overcome for forty-eight hours. The hypotension of the first twenty-four hours following operation may have been responsible for the transient urinary suppression. If this was a typical case of hepatorenal syndrome from the onset with renal insufficiency due to toxic substances of hepatic origin, how can one account for the urinary output of 930 and 1,030 cc. on the fourth and fifth postoperative days? The persistently elevated icteric index in spite of free biliary drainage, and the progressive decline in bile salts in the drainage bile speak for markedly impaired hepatic function. The rising NPN and urinary suppression are indicative of renal dysfunction, possibly functional at first but later associated with peripheral vascular failure, organic in nature. Whether some specific toxic substance finally broke down kidney reserve by direct effect, or whether the latter occurred secondary to persistent hypotension and histotoxic anoxia is a point for speculation. A normal temperature was obtained throughout the illness. Unlike Cases III and V, the vasomotor instability was not associated with tachycardia.

CASE VIII. M. S. M., a female, age sixty-two, developed jaundice and itching three days after an attack of gallbladder colic which recurred at intervals for two days. When admitted to the hospital the stools were clay colored, the icterus index was 97 but there had been no recent upper right quadrant pain. Hemoglobin was 74 per cent, red blood count 3,840,000, white blood count 7,000. The NPN was 34 mg. per cent, urea clearance slightly diminished to 60 per cent. An hippuric acid test of 23 per cent indicated marked liver insufficiency; however, a prothrombin time of 83 per cent was evidence against generalized liver damage. During five days of preoperative preparation for surgery there was no elevation of temperature and the patient remained free of distress. One hundred Gm. of glucose were given intravenously daily for three days before surgery. An hippuric acid test of 68 per cent the day before operation indicated a response to therapy.

Cholecystectomy and removal of a common duct stone with T-tube drainage of the common duct was done. The operation was tolerated well except for a transient fall in blood pressure to 94/70 with spontaneous return to 135/86. During the last twenty minutes of the operation 95 per cent-5 per cent oxygen carbon dioxide was given by mask.

On the evening of the day of operation the patient could not be roused for several hours following $\frac{1}{20}$ gr. dilaudid and $\frac{1}{200}$ gr. hyoscine. The color remained good, the pulse was 100 with an occasional dropped beat, the blood pressure 100/70 mm. Several hours later the effects of the sedative had worn off and the patient was easily aroused. Table VI shows fluid intake and output, temperature curve, blood chemistry determinations and the concentration of bile salts in the T-tube drainage. Eight hours after operation marked oliguria developed that persisted for forty hours. Between the sixteenth and twenty-fourth postoperative hours only 15 cc. of urine was excreted. During the second twenty-four-hour period the output was restricted to 60 cc., and the output for the third twenty-four-hour period was 66 cc.

On the morning of the second postoperative day the appearance was satisfactory, the patient was quiet, the temperature was 100.8°F., the blood pressure 140/86, the pulse 100 and regular. The bile drainage was scanty and dark containing only 120 mg. per cent of cholates. Prothrombin had diminished to 60 per cent, also indicative of suppressed liver function. The blood NPN was elevated to 58 mg. per cent. Decholin sodium was added to the intravenous glucose daily in quantities shown in Table VI. On the afternoon of the second day the patient appeared catatonic, staring at the ceiling and totally ignoring the examiner. The upper extremities were spastic but the reflexes were

equal and no pathologic reflexes were elicited. Later the appearance was more comatose, the breathing deep and labored, the blood pressure 140/90, the pulse 112 and regular.

The patient had been anuric for twelve hours. Continuous gastric lavage was started.

There developed a progressively deepening coma with muscular twitching and carpopedal spasm at intervals. Cheyne-Stokes respiration developed and there was some irregularity of the heart beat. Respiration ceased two minutes before the heart stopped.

TABLE VI

CASE VIII. M. S. M.

Days Pre- and Postoperative	5	3	2	1	0	1	2	3	4	5	6	7	8	9
Temperature	98^6	98^6	98^6	98^6	98^6	100^0	100^7	100^0	99^0	100^0	101^0	101^8	102^2	102^0
NPN mg, %	34						58	76					100	
Urea clearance %	60													
Hippuric acid %	23			68										
Prothrombin %	83			85			60		23			85		
BSP retention, 15 min. %					0							25		
Cholates in drainage bile mg. %					520	200	120	104	40	540	840	130	20	trace
Fluid intake, 24 hr./cc					4000	3500	4200	2500	3750	2000	3000	4500	2500	1000
Urine output 24 hr./cc					280	60	66	450	360	220	360	430	160	60
Parenteral glucose, Gm.		100	100	100	300	350	400	250	250	300	200	400	250	100
Liver extract* 10 cc. (H)								2	2					
Vitamin K, hypo										1	1	1	1	
Decholin, 20%, 10 cc							2	1	1	1	1	1	1	

* Lilly, crude.

On the morning of the third day there was no appreciable change in the general condition. Several ounces of urine were obtained for the night and during the twenty-four-hour period the output rose to 450 cc. The blood NPN was 76 mg. per cent. By the fourth day the concentration of cholates in the bile (400 cc. of bile for twenty-four hours) was reduced to the critically low level of 40 mg. per cent, the prothrombin had dropped to 23 per cent and the icterus had increased (blood bilirubin 10.2 mg. per cent in contrast to 7.6 mg. per cent before operation). The gastric drainage contained coffee-ground material. A total of 20 cc. of liver extract was given intramuscularly on the third and fourth days. Vitamin K (hyquinone) was given by hypo on the fifth, sixth, seventh and eighth days and the prothrombin time rose promptly from 23 to 85 per cent on the seventh day. On the fifth and sixth days the cholates in the bile drainage increased to 540 and 840 mg. per cent, respectively, only to drop off again to 130 and 20 mg. on the seventh and eighth days. A bromsulfalein test on the seventh day showed 25 per cent retention in fifteen minutes and the blood amylase was elevated to 304. On the eighth day the blood NPN was 100 mg. per cent and the urea N 56 mg. per cent. (Table VI.)

Comment. Although renal and hepatic function tests gave evidence of subnormal function before operation, clinically this patient appeared a fairly good operative risk. Urinary suppression developed eight hours after operation. The oliguria was associated with definite hepatic insufficiency as reflected by the increasing icterus, the drop in prothrombin and the marked impairment of bile salt synthesis. Decholin sodium was without recognizable effect on the course of subnormal liver function. It may have been a factor in the increased urinary output which began twenty-four hours after beginning this therapy.

The increased concentration of bile salts in the T-tube drainage on the fifth and sixth days following parenteral liver extract denoted greatly improved hepatic synthesis as did the response in prothrombin formation following parenteral vitamin K. (Table VI.) Despite these favorable signs, the patient did not show any clinical improvement. The catatonic stupor and the manifestations of alternating stimulation and depression of the central nervous system was out of all proportion to the degree

of azotemia present. The postoperative temperature curve is very suggestive of an associated infection. Permission for autopsy was denied.

Case ix. M. S., a female, age fifty-three, entered the hospital with a history of recurrent

the gallbladder after dye showed ·a poorly visualizing gallbladder with negative shadows.

Preparation for surgery for one week included in addition to a high caloric, high carbohydrate diet, vitamin B complex, bile salts and pancreatin. Atropine sulfate orally and luminal kept the patient free of discomfort.

TABLE VII
CASE IX. M. S.

Days Pre- and Postoperative	6	3	0	1	2	3	4	5	6	7	8	9	10	12	13
Temperature	98^4	98^6	98^8	101^0	100^0	99^0	99^8	100^0	98^6	99^8	100^0	100^2	100^0	100^0	99^4
NPN, mg. %	42	...	Opera-tion	....	85	83	50	48	....	....	46	....	....	....	32
Urea clearance, %	62	...		....	....	....	20		....	....		....	....	....	50
Hippuric acid test, %	..	121		....	....	....	50		....	....		....	....	....	80
Prothrombin, %	91	...	....	....	66	60	....		....	....	89		....	....	
Icteric index, units	8	...	....	....	.-...	60	....		....	....		....	....	....	10
BSP test, % retention	0	...	....	....	20	....		....	....		....	....		0	
24 hr. fluid intake cc	..	...	3000	4500	4200	1200	4300	3500	3500	3800	2000	150	2250	2000	2250
24 hr. urine output cc	..	...	500	340	90	210	1230	2250	2000	1170	940	1000	600	480	1260
Decholin, 20%, 10 cc	..	...	....	1	1	1	1	1	1						
Parenteral glucose, Gm	..	...	200	200	300	400	250	250	250	50	50				

attacks of severe colicky upper right quadrant pain with back and right shoulder radiation for twenty-five years. The initial attack in 1916 was described as severe. Between 1916 and 1939 the distress was milder in character, occurring at irregular intervals. During the past two years there had been an almost constant dull aching pain under the right costal border radiating at times to the right shoulder and to the epigastrium. There had been no jaundice, no fever or chills or weight loss. There was a selective dyspepsia for fried and "rich" foods.

Inventory by symptoms and past history were essentially negative except for the digestive disturbance. The patient had scarlet fever as a child. There had been four term pregnancies with normal deliveries and menopause in 1938.

Physical examination revealed a rather thin, fairly well nourished middle aged female with no remarkable physical findings except upper right quadrant tenderness. The blood pressure was 142/86, the pulse 88 and regular. Urinalysis was negative, hemoglobin 83 per cent, red blood count 4,060,000, white blood count 5,450 with a relative lymphocystosis. X-ray of

One hundred Gm. of glucose with 10 cc. of decholin sodium were given daily for five days before operation.

Tests of liver function were all normal before surgery (Table vii); hippuric acid test 121 per cent, prothrombin time 91 per cent, bromsulfalein test 0 retention in thirty minutes. Urea clearance (62 per cent) was slightly subnormal and a mild degree of nitrogenous retention was present on admission (NPN 42 mg. per cent, urea N. 24 mg. per cent).

Cholecystectomy under spinal anesthesia was uneventful except for a transient fall in blood pressure to 70/60 mm. Inhalation of 95 per cent oxygen—5 per cent carbon dioxide and 0.5 cc. adrenalin subcutaneously, promptly restored the pressure where it remained between 120 and 130 mm. The liver appeared grossly normal. The gallbladder was distended with clear bile and contained many variable sized stones.

During the first twenty-four hours after operation the course was not unusual except for diminished secretion of urine. (Table vii.) On the day following surgery continuous gastric drainage was begun because of nausea and frequent emesis. Despite a parenteral intake of

4,500 cc. of fluids containing 200 Gm. of glucose the oliguria persisted. Thirty-six hours after operation the sclera had a definite icteric tint and the patient looked ill. The blood pressure was sustained at its original level.

Blood chemistry and function tests forty-eight hours after operation indicated appreciable impairment of both liver and kidney function. The NPN was 85 mg. per cent, there was 20 per cent retention of dye in thirty minutes with the bromsulfalein test and the prothrombin time had dropped from the preoperative level of 91 to 66 per cent. Clinically the patient looked haggard and complained of nausea and weakness. The abdomen was flat and the gastric drainage bile stained. The pulse rate was 120, quality poor and the blood pressure 92/70 mm. A mild cyanosis and icterus were present on the evening of the second postoperative day when oxygen therapy was instituted (6 liters per minute by nasal catheter).

During the second postoperative day 4,200 cc. of parenteral fluids, 300 Gm. of glucose and 10 cc. decholin sodium were administered. The twenty-four-hour urine output was 90 cc. The patient appeared critically ill.

On the third postoperative day the appearance was somewhat improved. Oxygen was discontinued. The blood pressure was 160/95, the pulse 116 and of good quality, the temperature 100.4°F. The blood NPN was 83 mg. per cent, urea N 60 mg. per cent, prothrombin 60 per cent, icteric index 60. One ampule of decholin sodium and hypertonic glucose (400 Gm. in 1,200 cc. of fluid) was given in small infusions throughout the day in the hope of breaking through the urinary suppression. The urinary output for the twenty-four hour period was 210 cc.

On the fourth postoperative day the patient was extremely restless and confused at intervals. The blood pressure was 154/86, the pulse 116 and regular, and the temperature 99.2°F. An intravenous hippuric acid test was 50 per cent of normal in contrast to 121 per cent preoperatively. Despite the oliguria the NPN had dropped to 50 mg. per cent. Forty-three hundred cc. of parenteral fluid containing 250 Gm. of glucose with 10 cc. decholin sodium resulted in a twenty-four-hour urine volume of 1,230 cc.

On the fifth and sixth days active diuresis was obtained on an intake of 3,500 cc. containing 250 Gm. of glucose and 10 cc. decholin

sodium. (Table VII.) Gastric lavage was discontinued. From this point on the patient made an uneventful recovery. By the eighth postoperative day the prothrombin time had risen to 89 per cent without vitamin K therapy. On the thirteenth day a hippuric acid test was 80 per cent, there was no retention of dye in thirty minutes with the B.S.P. test, the skin had cleared of the jaundice and the blood NPN was normal.

Comment. This patient, as the others in group III, presented herself for surgery with kidneys having impaired reserve. The laboratory evidence of impaired hepatic function after surgery was more evident than in Case VIII, i.e., jaundice, lowered prothrombin, impaired removal of bromsulfalein dye as well as a lowered hippuric acid synthesis. However, impaired renal function invalidates the hippuric test in this instance. After better than sixty hours of pronounced urinary suppression a diuresis occurred following hypertonic glucose and decholin therapy. It is interesting and perhaps significant that the azotemia decreased in the presence of persistent oliguria. The fall in blood NPN and urea N preceded the increased urine output by a number of hours. It is to be noted that the blood NPN reached its highest level forty-eight hours after operation where it remained twenty-four hours despite a urine volume of 90 cc. for that period. Concomitant with the improvement in kidney function and clinical appearance the prothrombin value increased promptly without specific therapy, indicating rapid recovery of this particular hepatic function. The character of the temperature curve (Table VII) is suggestive of an associated mild infectious process of undetermined origin.

CASE X. H. G., a female, age sixty-six, entered the hospital with a typical history of intermittent biliary colic of ten years' duration. At onset attacks occurred once or twice a year but during the past two years they had become progressively more frequent so that during the past six months they occurred at weekly or

more frequent intervals. Of late morphine by hypodermic had been given for distress. There had never been associated jaundice, fever or chills. The attacks occurred at any time of the day or night.

Inventory by systems was non-contributory. A pelvic abscess had been drained in 1922.

Cholecystectomy was done under spinal anesthesia. The gallbladder was thick-walled, adherent to the liver bed and contained thick white bile and many faceted stones. The liver appeared normal. Except for a transient fall in blood pressure to 80/60 mm. which promptly returned to normal after 0.5 cc. adrenalin by

TABLE VIII

CASE X. H. G.

Days Pre- and Postoperative	5	4	3	2	1	0	1	2	3	4	5	6	7	8
Temperature	98^6	98^8	98^8	98^6	98^6	98^6	98^6	99^3	99^8	99^0	98^8	98^8	98^6	98^6
NPN, mg. %	48	..	40	...	36	Operation	....	60	54	46	50	....	42	38
Urea clearance, %	60	..	...	...	...		....	25	....	....	....	....	....	46
Hippuric acid test, %	..	74	...	...	86		....	30	....	....	....	....	....	58
BSP test, %	o	..	...	...	...		....	20	....	....	....	....	....	o
Prothrombin, %	86	..	...	...	...		....	83	....	....	....	....		
24 hr. fluid intake, cc	...	..	...	...	...	2500	2600	3500	2000	1500	1500	1800	1750	2000
24 hr. urine output, cc	..	..	...	...	...	1100	380	400	740	900	600	950	720	630
Decholin, 20%, 10 cc	..	..	...	...	...	1	1	1	1	1	1			
Parenteral glucose, Gm	..	.·.	100	100	100	200	250	300	150	150	150			

There had been five term pregnancies with normal deliveries and menopause in 1929 had been uneventful.

Physical examination revealed a well preserved, well developed, moderately obese woman in no apparent distress and without jaundice. Physical findings were essentially negative except for moderate tenderness in the upper right quadrant. The blood pressure was 166/84 mm., pulse rate 92 and regular in rate and force.

X-ray after dye revealed a non-visualizing gallbladder.

The patient was kept free of distress on atropine, nitroglycerin and two decholin tablets three times a day. Three days before surgery infusions of 500 cc. of 20 per cent glucose with 1 Gm. of calcium gluconate was given daily.

Laboratory work-up (Table VIII) revealed a slightly elevated NPN and urea N that declined to normal after preparation for operation. The hippuric acid test was subnormal (74 per cent), the urea clearance 10 per cent below the lower limits of normal (60 per cent) and there was no retention of dye at thirty minutes with the bromsulfalein test. The prothrombin time was 86 per cent. Following preparation for operation the hippuric acid test was 86 per cent and the blood NPN was within normal range.

hyperdermic and inhalation of 95 per cent oxygen-5 per cent carbon diozide, the operation was well tolerated. Six hours after operation the blood pressure was 178/110, the pulse 116, regular and of good quality. During the first twenty-four-hour period following operation 2,500 cc. of fluid was given parenterally containing 200 Gm. of glucose and 10 cc. of decholin sodium. A twenty-four-hour urinary output of 1,000 cc. was considered adequate. The same therapy was repeated during the second twenty-four-hour period except 250 Gm. of glucose were given. Urinary suppression was eminent with a twenty-four-hour output of 380 cc. During the afternoon the patient complained of nausea and fatigue and after several emesis of foul, dark material continuous gastric lavage was instituted. The temperature was normal, the blood pressure 164/88, the pulse 108 and regular.

On the second postoperative day the patient appeared listless but was in no distress. Laboratory studies gave evidence of impaired function of both liver and kidneys: intravenous hippuric acid test 30 per cent, bromsulfalein retention 20 per cent at thirty minutes, prothrombin 83 per cent, urea clearance 25 per cent, NPN 60 mg. per cent, urea N 58 mg. per cent. Thirty-five hundred cc. of parenteral fluid containing 300 Gm. of glucose and 10 cc. of decholin sodium was accompanied by a mild diuresis, the

twenty-four-hour urinary output reaching 1,400 cc. On the evening of the second postoperative day auricular fibrillation developed. Blood pressure was 154/56 mm., apex rate 156, radial pulse rate 96. The gastric drainage contained bloody fluid.

The same line of therapy except for lowered fluid intake because of the cardiovascular status was continued (Table VIII) throughout the third, fourth and fifth postoperative days with progressive clinical improvement. The heart rate became regular fourteen hours after the onset of fibrillation and gastric drainage was discontinued on the evening of the third postoperative day. With the twenty-four-hour urinary output between 600 and 950 cc. the nitrogenous retention decreased progressively to normal values on the eighth postoperative day. At this time the hippuric acid test and urea clearance were still impaired but were well above the values obtained forty-eight hours after operation. The patient was dismissed on the fifteenth postoperative day.

Comment. Any resemblance to the hepatorenal syndrome in this patient following cholecystectomy seems primarily due to poor kidney function. The drop in hippuric acid excretion cannot be ascribed to liver insufficiency because of the associated decline in urea clearance. We have seen normal hippuric acid excretion in patients in whom urea clearance was decreased 40 to 50 per cent of normal; however, below these values impaired kidney function invalidates the hippuric acid test as a reliable criterion of liver function. The retention of bromsulfalein, however, is indicative of definite hepatic dysfunction, although the prothrombin value of 83 per cent is against any degree of markedly impaired liver function. It is noteworthy in this case that following the onset of auricular fibrillation which contraindicated large volumes of intravenous fluids, the nonprotein nitrogen dropped to normal values despite the fact that urinary output remained at levels which are supposedly inadequate to clear the blood of nitrogenous waste products in the presence of impaired kidney function. Decholin sodium therapy may possibly have influenced the existing azotemia by some unknown effect on urea and nitrogen metabolism.

COMMENTS

Vital organs, of which the liver and kidneys are examples, are characterized physiologically by large functional reserves—large factors of safety. It has been estimated that but one-fourth of the parenchyma of these organs functioning *normally* is sufficient to carry out bodily functions. Decompensation follows when functional reserves are exhausted. Laboratory tests fall into two categories: *function tests*, such as the hippuric acid and urea clearance tests, and *decompensation tests*, such as determinations of nitrogenous retention or the quantitation of icterus. The former give us insight to the state of functional reserve. The latter give an idea of severity of parenchymal damage in an organ whose functional reserve has been depleted. There is no justification for the assumption that liver and kidney function are "normal" in a patient who is not jaundiced and whose NPN is within normal limits. This situation obtains not infrequently in patients who are without functional reserves and who are thrown immediately into decompensation by insult that would ordinarily be considered insignificant. Yet much of our present knowledge of the hepatorenal syndrome springs from such a premise.

Considerable supportive evidence of a postoperative liver-kidney relationship is indirect. In many instances conclusions have been reached by *a priori* interpretation of hospital records and necropsy material. The functional state of the organs under indictment prior to the onset of pathologic decompensation was problematical or unknown. This is particularly true of kidney function. Bartlett[2] has called attention to this shortcoming. Before postoperative renal insufficiency can be attributed to extrinsic origin appropriate tests which demonstrate previously existing normal renal reserve must be on record.

There is a paucity of such data in hepatorenal literature.

Certain clinical and laboratory findings were so prevalent in our group of patients that they deserve evaluation as potential forerunners of the so-called hepatorenal picture in surgical patients.

Compensated Renal Insufficiency before Surgery. Nine of our ten subjects had a normal NPN before surgery. However, eight of the ten possessed varying degrees of diminished renal reserve which was revealed by subnormal urea clearance. Obviously, the development of a hepatorenal picture in these patients stemmed from kidneys with impoverished reserves.

Vasomotor Instability. Anoxia during or following surgery is an important predisposing factor in the hepatorenal picture. Seven of the ten patients in our series developed delayed postoperative hypotension. Five of the seven had previously been exposed to transient fall in blood pressure to shock levels while on the operating table. This followed, frequently but not always, traction on the mesentery or biliary tract. Only two of our subjects experienced a drop in blood pressure during the operation without a recurrence of hypotension during the postoperative period. This is significant. In all instances except Case VII the blood pressure was restored during the operation by subcutaneously administered adrenalin and 95 per cent oxygen-5 per cent carbon dioxide by mask. Four of the six patients developed hypotension, 70 mm. systolic or less, within twelve hours. In the remaining two, vasomotor collapse was delayed twenty-four and forty-eight hours, respectively.

We have observed significant drops in hepatic function in healthy surgical patients following transient shock during operation.[28] The poor risk patient with limited hepatic and renal reserves tolerates a like degree of anoxia even more poorly if at all.

Factors other than anoxia may be at work in patients who exhibit such a tendency to vasomotor collapse. Boyce[5] suggests that such reactions may be anaphylactoid in nature: following operation there is released by the liver a hypothetical toxic substance to which the patient has become sensitized. Anaphylactoid shock follows. Only indirect experimental evidence supports such a view. Based on more concrete experimental evidence is Ravdin's vasodepressor theory.[25] This investigator has extracted vasodepressor substances from the livers of dogs with obstructed common ducts. An autoclaved, alcoholic extract of fresh normal liver produced less of a drop in blood pressure (anesthetized cat) than a comparable dose of jaundiced liver extract. A dose of extract representing as little as 40 mg. of jaundiced liver substance produced a fall in pressure. Obstructed livers contained more histamine and choline than normal livers. Working along a similar line of investigation, Boyce[5] extracted the livers of patients whose postoperative clinical course and microscopic sections of liver and kidney conformed with the hepatorenal picture. Dogs injected with saline and watery extracts showed parenchymatous degeneration of the liver cells with comparable changes in the convoluted tubules of the kidneys. At autopsy a dog injected with alcoholic liver extract showed no significant findings. Neither blood pressure determinations or blood chemistry studies were reported in the latter's studies. Presumably the two investigators were dealing with different liver fractions inasmuch as Ravdin's vasodepressor substances were alcohol soluble while Boyce obtained positive results only with saline and aqueous pathologic liver extracts.

Early Hepatic Insufficiency, Urinary Suppression and Azotemia. Nine of ten patients gave evidence of decreased liver function within twenty-four to forty-eight hours following operation. However, the earliest sign of impending trouble was urinary suppression despite adequate fluid intake. As a rule, urinary suppression became most marked during the second twenty-four-hour postoperative period al-

though in half of the cases oliguria developed within the first twelve hours. Because of impaired renal clearance results of the postoperative hippuric acid test were considered invalid. Prothrombin time, dye elimination, icteric index or blood bilirubin levels, and in two instances, bile salt concentration in drainage bile were used as liver function criteria.

It is our impression that the secretory activity of the kidney is compromised earlier and to a greater degree than hepatic function during the same interval. In three subjects who recovered from what appeared clinically as a critical, fully developed hepatorenal picture (Cases IV, IX and X) the onset of diuresis was followed by clinical improvement. Curiously, patients IV and IX appeared to have passed the crisis and the blood NPN had begun to decrease *six hours before* active diuresis was an actuality. On the contrary, improved renal elimination in patients I, II, XVII and VIII was without effect on the fatal course. In the remaining three subjects oliguria and azotemia were progressive.

The ability of the liver to respond to specific therapy in one or more of its functions after the hepatorenal picture is well developed was surprising and is worthy of comment. Patients with subnormal hepatic function who respond poorly to specific therapeutic measures directed at improving liver function tolerate operative procedures much more poorly than patients who, although poor risks, show improved liver function after the same preparation.

CONCLUSIONS

1. The liver plays an important rôle in postoperative morbidity and mortality. Patients with evidence of subnormal liver function who responded poorly to specific measures directed at improving hepatic function were poorer surgical risks than those who responded favorably.

2. Patients who developed hepatic and renal insufficiency following surgery usually gave evidence of subnormal renal reserve prior to operation.

3. Diminished renal reserve plays a greater rôle in the development of the hepatorenal syndrome in surgical patients than is generally recognized. Knowledge of the blood NPN is without information on renal reserve until such reserves have been depleted.

4. Transitory fall of blood pressure and anoxia in poor risk surgical patients should be anticipated during and after surgery. Preventative treatment is the most effective treatment of circulatory collapse.

5. Decholin sodium appears to have produced favorable results in the treatment of postoperative hepatic and renal insufficiency.

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DOUBLE FRACTURES AND DOUBLE NON-UNIONS
OF THE SHAFT OF THE TIBIA

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DOUBLE fractures of the shaft of the tibia are not infrequent. Their healing process is slow and the incidence of non-union high. The cause of it is the disturbance of the intra-osseous circulation following such fractures.

Anatomically, there are two vascular systems in the normal long medullated bone (Langer, Lexer, Delkes-Kamp and Kolodny,) the periosteal and the intra-osseous. The periosteal system consists of a net of smaller and middle-sized vessels which are derived directly from the surrounding soft tissue and are in intimate connection with them. Only a few small anastomoses connect the periosteal with the intra-osseous circulation. The intra-osseous system consists of three sets of vessels (Fig. 1): The first set is that of the diaphysis containing the nutrient artery which enters the bone as a single trunk except at the femur when it is double. (Fig. 1a.) Very soon it splits up into an ascending and descending branch and crosses toward the metaphysis. The second set is formed by the metaphyseal vessels (Fig. 1b) which are branches of the vessels of the joint capsule. They penetrate the bone near the insertion of the capsule beneath the epiphyseal line and supply the metaphysis. The third set is that of the epiphysis (Fig. 1c), the vessels of which are derived from the joint ligaments. In youth the three intra-osseous sets are connected only with a few anastomoses. With growth the anastomoses become more numerous, and after the period of development the three sets form one system.

From these considerations it becomes evident that the periosteal and intra-osseous system have only a few anasto-moses. Hence the bone itself is chiefly vascularized by the intra-osseous system. In an ordinary single fracture of the shaft of the tibia, both fragments remain well vascularized even if the fracture should happen to be through the entrance of the nutrient artery, in other words, in level of the foramen nutricium. (Fig. 2a.) The collateral circulation from the metaphyseal vessels is sufficient to assure adequate blood supply to both fragments. In a double fracture of the shaft of the tibia, however, the middle fragment may become deprived of the bulk of its blood supply (Fig. 2b) unless it contains the nutrient artery.

As a rule, in the human tibia the nutrient artery enters the shaft within the proximal third at the posterior surface near the linea poplitea. Hence, in a double fracture in which the proximal fracture line runs through the foramen nutricium or distal to it, the middle fragment is apt to become deprived of its blood supply and may become necrotic. If this is the case, the incidence of non-union is high. The middle fragment can become regenerated only by ingrowth of vessels from the proximal and distal fragments and from the surrounding periosteum. This process is slow and non-union may develop in the meantime. In such a case the best treatment is complete and prolonged immobilization of the limb in a skin-tight plaster cast to promote and not to disturb the ingrowth of vessels from the peripheral fragments. If the proximal fracture line runs proximal to the foramen nutricium, the central fragment remains vascularized unless the nutrient artery becomes injured from the trauma and displacement of the fragment. If it remains vascularized, the healing

FIG. 2. *a*, Experimental fracture of the radius of a dog three weeks after the fracture. The fracture line runs just distal to the entrance of the nutrient artery. Both fragments are well vascularized, the proximal fragment from the descending branch of the nutrient artery and the distal fragment from the collateral circulation from metaphyseal vessels. *b*, Double fracture where the fracture lines would run through the entrance of the nutrient artery and the second one distal to it; the middle fragment would be deprived of the interosseous circulation.

FIG. 1. Representation of the bone vessels of the radius of a dog. The dog was killed under anesthesia. Immediately afterward the shoulder girdle was disarticulated and the vessels injected with a turpentine mercury solution through the arterial axillaris. The radius was removed. The periosteum was scraped from the bone and an x-ray picture was taken. The distal part (DME) is still in the stage of growth. Hence, the vascular systems are still separated from each other. The proximal part (*dme*) is beyond the period of development. The vessels have undergone anastomosis. *a*, Entrance of the nutrient artery; *b*, entrance of the metaphyseal artery; *c*, entrance of the epiphyseal artery.

Fig. 3. A, double non-union of the tibia seven months after the accident. The middle fragment is apparently deprived of the interosseous circulation and has undergone aseptic necrosis as evidenced from the density of the fragment. (Compare with Fig. 2b.) B, the shaft of the fibula was transplanted to the tibia bridging both non-unions.

process of the fracture will take the ordinary course.

From roentgenologic studies alone it is difficult to judge whether the proximal fracture line runs above or below the entrance of the nutrient artery since only rarely would it be possible to visualize the foramen nutricium. Hence it is safer to treat a double fracture of the shaft of the tibia rather by complete immobilization than by traction.

Later studies, however, are of value concerning the changes of the bone structures. If the middle fragment does not atrophy in the same degree as the proximal and distal fragments do, in other words, if it remains dense, it must be assumed that it is deprived of its circulation which otherwise would permit the calcium to be carried away. (Fig. 3A.) If non-union has developed, obviously the best way to treat it is by bone grafting. The bone graft, however, should be long enough to bridge both non-unions. This would require a long massive graft from the tibia of the other side. This, however, may weaken the other leg. To avoid this the author advises the use of a fibula graft taken from the same side.

CASE REPORTS

CASE I. A patient, aged twenty-one, sustained a fracture of the right fibula and a double fracture of the right tibia in a motorcycle accident. The fibula fracture healed; the double fracture of the tibia had not healed seven months after the accident. Note in the illustration the density of the middle fragment as compared with the atrophy of the proximal and distal fragments. Hence from x-ray examination it was evident that the middle fragment was dead. To promote healing of the non-

union bone graft operation was performed. The tibia was exposed from an anterior incision; the middle fragment was removed subperiosteally. It then was halved and the posterior half was replaced. The proximal and distal fragments were prepared for reeeption of the bone graft, i.e., the anterior cortex was removed. From a lateroposterior incision the fibula of the same side covered with its periosteum was removed. It was laid upon the three fragments and fastened with wires. An x-ray taken five months after the operation showed satisfactory healing conditions. The patient was discharged in a walking brace. About a year after the operation the non-union cleft became less visible and the middle fragment organized. In order not to delay regeneration the wires were removed. Two years after the operation, graft and host bones had become an organic union. The patient walked without support or a limp. (Figs 3A to C.)

Grafting of the fibula or of a large massive bone graft is an extensive operation. If the patient's general condition is such as to make this procedure unsafe, the writer recommends the transfer of the fibula to the tibia of the same side in stages. Hahn (1884) was the first to conceive the idea of fibular transfer. He was followed by Codivilla. The method was subsequently improved by Huntington, Stone, Campbell, Wilson and Myerding. The technic varies with the age of the patient. In adults the author prefers fusion of the upper tibiofibular joint in the first stage (Campbell, Meyerding), and an osteotomy of the lower part of the fibula and fusion of this part with the tibia (Wilson) in the second stage. The method has many advantages: the fibula does not become separated from its blood supply; the transferred and fused fibula provides a solid strut and it also permits immobilization of the tibia so that non-unions may heal even without subsequent bone grafting.

CASE II. A patient, aged fifty-two, was struck by an automobile. He was admitted unconscious with fractures of the right clavicle, right radius, eighth rib, right fibula and double fracture of the right tibia. He regained consciousness soon. His general condition re-

FIG. 3. C, two years after the operation graft and host bone have become an organic unit.

mained precarious. No treatment was required for the fractured clavicle; fracture of the radius was reduced and immobilized; a skin-tight plaster cast was applied to the right leg from his toes to the mid-thigh. All fractures healed with the exception of the double fracture of the tibia. The middle fragment became dense while the proximal and distal fragments underwent atrophy. These x-ray findings made it evident that the middle fragment was deprived of adequate circulation and had undergone aseptic necrosis. A major bone grafting operation did not appear advisable since the general condition of the patient remained impaired. Hence a fibula transfer in two stages was carried out four months after the accident. (Figs. 4A and B.) The first stage included fusion of the upper tibiofibular joint. From an incision parallel to the peroneal nerve the latter was located, dissected free and held away with a tape. The head of the fibula was now freed from its ligaments. The opposing surfaces of the tibiofibular joint were thoroughly denuded by removing cartilage
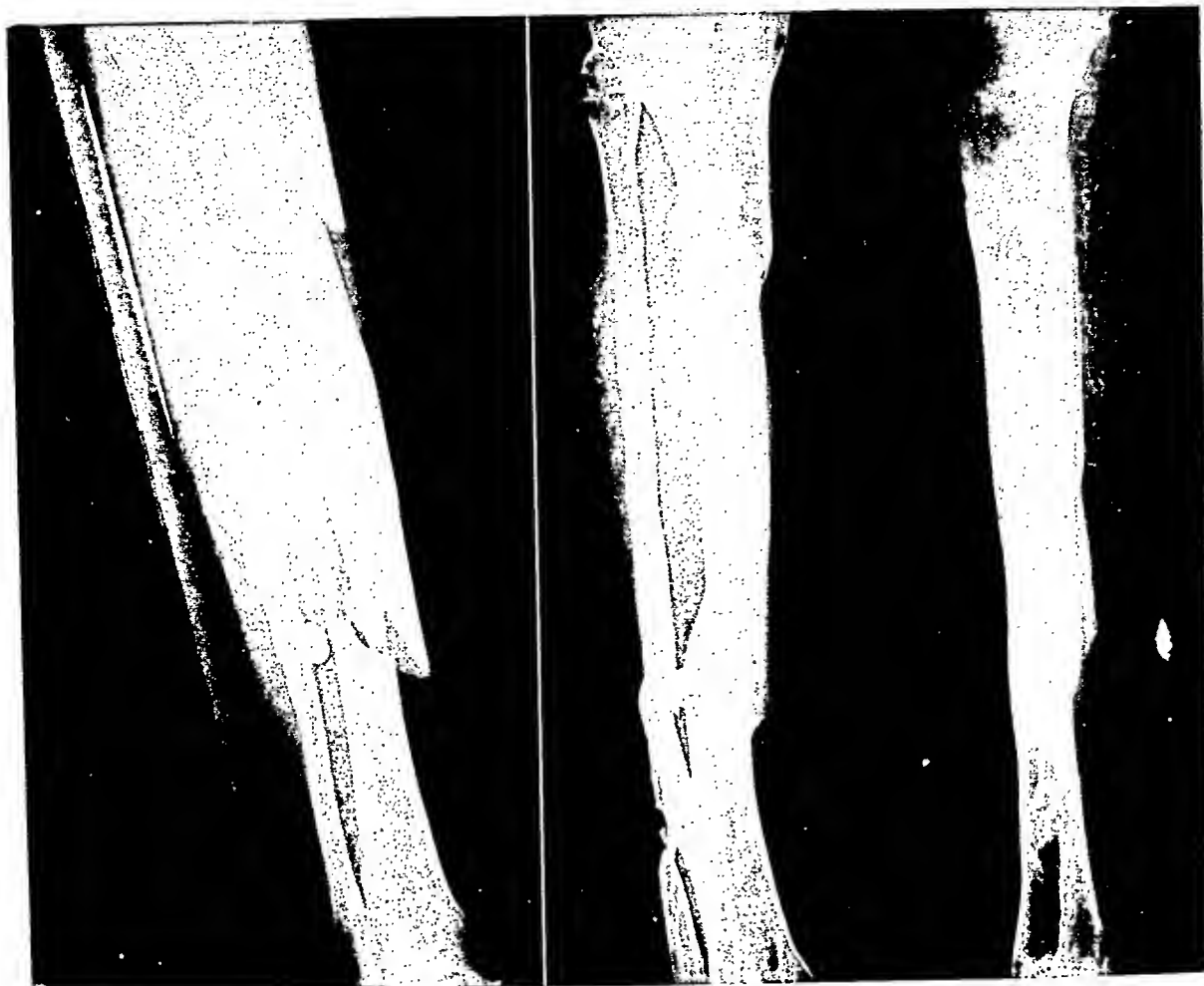
FIG. 4. A, double non-union of the tibia four months after the accident. B, fistula transferred in two stages. First stage, fusion of upper tibiofibular joint (note bone peg through head of fibula into the tibia); second stage, six weeks later osteotomy between middle and distal third of fibula and fusion of proximal fragment of fibula to tibia. This roentgenogram was taken one year after the first operation.

and periosteum with chisel and curette. A hole was excavated in the tibia opposite to the head of the fibula until the median surface of the head of the fibula could be almost inserted into the tibia. To obtain better union a canal was drilled through the head of the fibula and tibia and a bone peg was inserted which was removed from the upper anterior crest of the tibia. I recommended this since it provides a firmer internal fixation and the autogenous bone graft promotes quicker and firmer fusion. The second stage was undertaken six weeks later. With a tourniquet applied the ankle joint was exposed from an anterior incision between the tibia and fibula. The underlying tendons and muscles were retracted on either side until tibia and fibula were clearly visible. The fibula was now osteotomized with a chisel

about 8 cm. above the ankle in an oblique plane from below inward to above outward. The lateral surface of the tibia was now exposed. With a blunt instrument, a hole was made in the interosseous membrane and the muscles anterior to it. The shaft of the fibula was now pulled through this hole and approximated to the tibia. At the point of approximation a flap of tibia cortex (the base being distal and lateral) was split. A groove was excavated medial to it for reception of the fibula. The fibula was now inserted into the prepared tibia. During this manoever the fibula fractured. After the fracture occurred the transfer of this part of the fibula was much facilitated. After closure of the wound a plaster cast was applied from the toes to the mid-thigh for a period of one month. It then was removed and replaced by a cast in

which a walking iron was incorporated. This cast was removed after two months. Swelling of the leg from poor circulation (arteriosclerotic changes) subsided gradually upon physiotherapy. The patient walked on crutches.

Eight months after the last operation the patient fell and fractured the femur of the same side through the intertrochanteric line. Insertion of a Smith-Petersen nail was performed according to the Engel-May method with application of an intertrochanteric attachment. Seven days after this operation the patient fell out of his wheel chair upon his right leg. All fractures held. He was up and around on crutches thirteen days after the operation. He walked without any support ten months after the second operation and two months after the nailing operation. The nonunions healed.

SUMMARY

The cause of the slow healing and the high incidence of non-unions of double fractures of the shaft of the tibia is due to the disturbance of the interosseous circulation following such fractures. If a double non-union develops, transplantation of the fibula of the same side as the bone graft is recommended, or the transfer of the fibula of the same side to the tibia in stages. In the first stage the upper tibiofibular joint is fused. To achieve firmer and quicker fusion insertion of a bone peg is recommended. In the second stage an osteotomy in the lower part of the fibula is performed and this part of the fibula is fused with the tibia.

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EXTRAPERITONEAL CESAREAN SECTION AS A PROCEDURE OF CHOICE

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A QUESTION that still remains in the minds of many obstetricians today is whether a true extraperitoneal operation is needed. Reductions in the mortality and morbidity of the various types of cesarean section have been so decided in the past twenty years that some men still question the superiority of the low transperitoneal retrovesical cervical operation when compared with the classical incisions (high or low). This may be true in the selective group of sections, but when one has to deal with frankly or potentially infected cases and with those in whom previous attempts at delivery have failed, all obstetricians agree that neither operation is effective in preventing peritoneal contamination. Despite tremendous experience, judgment as to whether craniotomy, the Porro operation or the extraperitoneal operation is the method of choice becomes difficult.

Each of these procedures has its advocates, and each advocate, I believe, is sincere in his conviction that he is doing what he thinks is right. Greenhill states, "In my experience the Porro operation is the safest one in the presence of definite infection." C. Buscoe finds, "the supposed protection given the infected patient by the Porro operation has been lacking in Philadelphia (1932–1944). Reports on extraperitoneal cesarean sections show the death rate from all cases to be from two to five times less than that from sepsis alone in cases in which the Porro operation was done." Monahan, Connally and Eastman believe, "that cervical cesarean section becomes increasingly hazardous after 18 hours of labor and that such cases had best be superseded either by cesarean hysterectomy or an extraperitoneal operation."

My experience with the Porro operation in frankly infected cases has been rather limited; but when I have had to resort to this operation, I have always removed the cervix as well and drained the pelvis. With the cervix allowed to remain, I can see no reason to defend the Porro operation in infected cases. Many series of cesarean hysterectomy can be cited with excellent results, but I question whether the showing would be any too impressive if these series were limited to infected cases only. Most of the Porro operations have been done for ruptured uteri, Couvelaire uteri, fibromyomas of the uterus, etc., in which intrauterine infections were absent.

Norton, Waters, Steele, Burns, Ricci and others prefer the extraperitoneal operation on the grounds that uteri are not removed needlessly and that babies are not sacrificed. Where there is probable or actual existence of intrauterine infection, they recommend the extraperitoneal operation. They claim that if the operation is properly performed postoperative peritonitis can be eliminated as a cause of death and that the uterus is conserved as well. To utilize this type of cesarean section in the very worst cases means that it must have virtues not obtainable in the transperitoneal operations. Some men (Henkel et al.) claim that the only feature in favor of the extraperitoneal section was that it prevented subsequent intra-abdominal adhesions. Sellheim and Kronig and in later years Beck and others believe that "careful walling off of the abdominal cavity with gauze was sufficient protection against infection."

Today we can truly dispute these statements. While the use of blood transfusions, sulfonamides, penicillin, streptomycin, etc.,

have made the lower segment two-flap type of cesarean section safer in those patients who have been in labor and those in whom the membranes have been ruptured for a period exceeding twenty-four hours, no conscientious obstetrician of today will admit that the intraperitoneal operation is as safe as the extraperitoneal types. The results to date with the extraperitoneal operations have been excellent but they do not tell the profession that it is a superior type of section. To do this one must be able to see what this operation can accomplish in large series of selective cases as well. Dr. H. Halsted, in discussing Waters's presentation of the "supravesical extraperitoneal cesarean section" before the New York Obstetrical Society in January, 1939, stated, "I feel with Dr. Burns that the extraperitoneal operation is an extremely valuable operation, that it is not done often enough, and I would like to see it developed, so that it can be used on patients who have not been in labor." Five years before this (1934) G. Kaboth reported on 308 extraperitoneal sections and considered it the least trying type of cesarean section for the mother and recommended it for all cases requiring delivery by abdominal section. Stimulated by Kaboth's report and Halsted's suggestion, the writer has performed the extraperitoneal operation as his routine method of section in elective cases to convince himself that the following statements were correct:

First. Any obstetrician with sound surgical background and training can overcome the technical difficulties entailed in performing the extraperitoneal operation. Stearns, after performing the Waters's supravesical operation in sixteen infected cases, opened the bladder inadvertently in three cases and invaded the peritoneal cavity in seven cases. He concluded, "This procedure should definitely relegate the Porro operation to its proper place in oblivion, save an occasional case." In my humble opinion, 43 per cent peritoneal perforations and 18.7 per cent bladder

injuries do not spell success in surgery even though no mothers were lost, nor does it help to advance the cause for extraperitoneal cesarean section. With such results, it might be wiser to relegate the extraperitoneal operation to its place in oblivion and resurrect the Porro operation. Stearns further stated, "Extraperitoneal cesarean section need not and should not supersede low cervical intraperitoneal cesarean section, for the technique is learned with some difficulty and is sufficiently complicated that the average obstetrician needs a certain amount of training to become proficient. There should be in every large community a few men trained in the procedure to salvage the occasional mother or child who cannot be delivered safely in any other way." Daichman and Pomerance reported their results with the Waters's supravesical type of operation in one hundred cases with no maternal deaths. Unfortunately again for the reputation of this excellent operation, they reported that in thirty-six cases, one or more openings in the peritoneum occurred; that, in their series seventeen bladders were injured, and that in one case vesicovaginal and vesico-abdominal fistulas occurred.

With such a high incidence of bladder and peritoneal injuries, it is not surprising that many obstetricians do not care to learn the extraperitoneal operations. While it is true that the extraperitoneal operation does carry certain real and vexing dangers to the peritoneum, bladder, ureters, and large vessels in the vesico-uterine space and broad ligaments, and that the occurrence of vesicovaginal fistulas is lamentable, nevertheless it has been the experience of many men now well acquainted with the extraperitoneal operation that these accidents were commoner in the earlier days of their experience and that later on these pitfalls were almost always avoided.

The author has had ample opportunity to try several extraperitoneal approaches to the retrovesical, infraperitoneal space and is convinced that the paravesical approach utilized, developed and advocated

by J. F. Norton has much to offer over the supravesical approach of Waters. It is less time consuming, is less apt to produce peritoneal or bladder perforations, and can be mastered by any well trained obstetrician. Regardless of which method is chosen, success or failure will depend upon a good knowledge of the fascial structures and their relationship to the lower uterine segment, the peritoneum and the bladder. The hazy early anatomical descriptions of the anatomists have been replaced by detailed descriptions of both the anatomy and the surgical technics by such present day contributors as Burns, Steele, Ricci, Marr, Waters, Norton, Cartwright, Furniss and others.

In 1920 Lichenstein noted "that the longer the patient was in labor, the less likelihood there was of injuring the peritoneum. This was found to be true only in cases in which the membranes had not ruptured. If the membranes had ruptured early, the os remained practically unopened and the cervix did not retract." He concluded, "that in these cases, the extraperitoneal operation is difficult." While the effacement and dilation of the cervix unquestionably cause the level at which the visceral peritoneum fuses with the uterus (i.e., the upper limit of the isthmus) to rise and thus facilitate the performance of the extraperitoneal operation, the writer does not believe that this factor makes the extraperitoneal operation technically more difficult for the skillful obstetrician; nor does his experience allow him to concede that the operation will be fraught with serious dangers to the ureters and bladder, or that the peritoneum will be unavoidably injured in many instances. Having performed extraperitoneal hysterotomy at four months and, in not a few instances, having performed the extraperitoneal operation in placenta previa in cases between six and eight months, the writer has come to the conclusion that this operation can be used freely as the procedure of choice in nearly all cases requiring cesarean section. In the writer's series of one hundred cases of extraperitoneal cesarean section, there were no injuries to the bladder, ureters or lateral pelvic veins. The peritoneum was button-holed in only three cases, an incidence of 3 per cent, and these were infinitesmally small. They were repaired in the manner recommended by Ricci and others by tenting the peritoneum. When one considers that in at least 20 per cent of the writer's cases previous low flap cervical sections had been performed, one must concede that a 3 per cent incidence of peritoneal perforations is extremely small. Furthermore, these three perforations occurred in the first fifteen cases of this series. In the last eighty-five cases no peritoneal rents were made.

It has been brought out by Norton, Waters and others that there may be limitations of the exposed extraperitoneal space in "deep pelves and fat women." The peritoneum in these obese patients is apt to be exceedingly thin and easily torn. With proper care, neither the deepness of the pelvis nor the thinness of the peritoneum should act as deterrents; rather they should caution us to greater delicateness in the handling of tissues. The presence of many basal retrovesical varicosities of considerable size can be seen, but these need not alarm the operator as the incision can frequently be placed so that these can be avoided. These can best be avoided by elevating the upper fascioperitoneal flap a little higher than usual. Those that must be incised can be easily controlled by Allis clamps. A word of warning at this point may not be out of place: Regardless of which approach is chosen, the operator should try to keep all of the dissection lateral to the lateral umbilical ligaments. He must not traumatize the area lateral to these ligaments or else some of the larger vesical veins or lateral pelvic veins will be injured. Bleeding here, while not alarming, can be troublesome. Performed with due consideration of these details, the operation can be kept almost bloodless.

Second. The operation is a safe one from the standpoint of morbidity and mortality. Shock is reduced to a minimum due to the fact that the intestines do not come in contact with the operative field. Since no intra-abdominal manipulations are

TABLE 1
ANALYSIS OF ONE HUNDRED CASES OF EXTRAPERITONEAL CESAREAN SECTION

Maternal Mortality......................	0
Maternal Morbidity (usual standards) (average).....................	0.5 days

No. Cases
  69........ no morbidity
  23........ one day morbidity
  6........ two days of morbidity
  2........ morbidity of three days or more (both due to wound infections)

No. of babies born alive...............	100
Fetal deaths.........................	6

  1. Placenta previa—7½ months
  2. Placenta previa—6½ months
  3. Placenta previa—7 months
  4. Severe pre-eclampsia—7 months
  5. Congenital defects—multiple
  6. Mongoloid idiot
     All Born Alive but Died within 72 Hours

Operating time average...............	30 min.
Wound infections....................	2
Days in hospital....................	9.12 days
No. drained.........................	3
No. in whom sulfonamides were used....	3
Peritoneal perforations...............	3
Bladder injuries.....................	0
Ureteral injuries....................	0
Injuries to lateral pelvic veins..........	0
Distention..........................	0
Difficulty in the delivery of babies......	0
Urinary complications................	0
Number of transfusions...............	0
Amount of bleeding..................	Normal
Thrombophlebitis and phlebothrombosis.	0

possible, postoperative distention and ileus are practically unknown. There should, therefore, be no postoperative adhesions. While postoperative adhesions are quite rare in the cervical two-flap type cesarean section, they do occur and act as a potential threat to the patient for the rest of her life. In the writer's series of one hundred cases shock never occurred. In no case was a transfusion necessary. The amount of blood lost never exceeded that seen in a normal delivery. There was not a single case of ileus or postoperative abdominal distention. (Table 1.)

Third. The delivery of a full term baby of normal or larger than normal size should be easily accomplished. In most of the reported series of extraperitoneal cesarean sections, delivery of the fetal head was accomplished by forceps or by a vectis or single blade. It is the writer's guess that in not a few cases this was responsible for injury to the peritoneum and bladder. With this in mind, the writer has developed a technic which, if followed, will render the delivery of the baby infinitely easier.

Version and breech extraction is performed routinely on all vertex cases and is practiced as follows: If the fetal back is located on the mother's left, the operator inserts his left hand through the cervical incision. The baby's forehead and face are grasped by the left hand and pushed in the direction of its chest to obtain the maximum flexion of the neck. The head is carried to the left iliac fossa and allowed to remain there. The external hand (right) then pushes the breech downward and to the right so that the feet can be grasped by the left (internal) hand of the operator. It is preferable to grasp both feet and this should always be possible. If only one foot is grasped, the posterior foot should be chosen. (If the anterior foot is grasped, the posterior leg may splint itself against the maternal spine during the extraction of the anterior leg.) Once delivered, the leg or legs are grasped by an assistant and extracted by traction toward the ceiling. Before the assistant exerts any traction the operator reinserts his hand (the right hand is now preferable), and gently pushes the head up into the fundus while the assistant pulls on the legs. Both movements should be synchronized. The legs are rotated and extracted so that their extensor surfaces face the mother's head. This allows the abdomen to present anteriorly (not back anterior as is the practice of many men in the delivery of breech cases through the vagina). Traction is now made upward toward the ceiling and the body of the baby is slightly rotated counter clockwise (45 degrees) until the anterior (left)

scapula of the baby is seen. In this position the baby's back is toward the mother's left and the baby's left or anterior scapula is presenting. The fetal legs and body are swung in the direction of the mother's right knee. This allows the left or anterior arm to be delivered with ease. To deliver the posterior arm, the fetal legs and body are now swung upward in the direction of the mother's left shoulder. This allows the right (posterior) arm to be delivered. Should the baby's head and back lie to the mother's right side, the same procedures are carried out only in opposite order: The delivery of the aftercoming head will be no problem if the body and head are now rotated through an arc of 135 degrees so that the baby's occiput comes anterior. With a finger in the baby's mouth, flexion is accomplished and maintained. Traction on the aftercoming head and body is not downward but upward toward the mother's head. This can be facilitated by downward pressure by an assistant's hand placed over the fundus of the uterus. Forceps to the aftercoming head have never been necessary. In not a single case of this series has the writer experienced the least difficulty in the extraction of the babies. When the above maneuvers are carried out, the ease with which the babies can be delivered is surprising.*

Fourth. The operation must not be too time consuming. While the success of most operations should not be measured by time, I believe that in cesarean section the shorter the time, the less the bleeding and shock and the smoother the convalescence. It was argued that the extraperitoneal operation was too time consuming to be practical. In the writer's cases the shortest time was eighteen minutes, the longest time forty minutes; the average time was thirty-five minutes when the supravesical approach was used and thirty minutes

* When spinal anesthesia is used and the uterus is firmly contracted, version and breech extraction may be a little more difficult; but it is still possible and is easier and safer to accomplish than extraction with forceps.

when the paravesical approach was used. The writer is now determined in his decision to continue the paravesical approach in all of his future cases.

Fifth. The operation can be performed with an empty bladder. Most operators distend the bladder at the beginning of the operation, empty it when the proper cleavage planes are found and refill it at the completion of the operation to test its integrity. It has been the writer's belief that this consumes time, exposes the bladder to increased risk of injury and makes dissection more difficult. In none of the writer's cases has the bladder been filled and in none of his cases has the bladder been injured.

Sixth. The operation should be possible as a repeat procedure in cases in which a test of labor is given in a pregnancy that follows a previous cesarean section. If the test of labor should prove a failure, an extraperitoneal cesarean section should still be possible. With this in mind, the writer has purposely resorted to an extraperitoneal operation in cases that had previous intraperitoneal two-flap cervical cesarean sections. It has been his finding that while the bladder is more firmly anchored to the lower uterine segment than normally, dissection was still possible without injuring either the peritoneum or the bladder.

CONCLUSIONS

1. After putting the extraperitoneal operation through a rigid test in one hundred cases, the writer has come to the conclusion that if the extraperitoneal operation is the safest operation for infected cases, it should be the safest in clean cases. The writer agrees completely with Kaboth that, "it is the least trying type of cesarean section for the mother," and, "that it can be recommended for all cases requiring abdominal section."

2. The dangers of postoperative hernia, even where drainage of the operative area is used, is reduced to a minimum.

3. The operation can and should be performed with an empty bladder.

4. The babies can be easily delivered by version and breech extraction. The use of forceps should be avoided.

5. There should be no postoperative adhesions or ileus.

6. Shock is reduced to a minimum since no intra-abdominal manipulations are necessary and no bowel comes in contact with the operative field.

7. Hemorrhage should be minimal.

8. There should be no danger of postoperative peritonitis.

9. There should be ease of performance which increases as experience is gained.

10. It can readily be used as a routine procedure in place of the intraperitoneal operation.

11. It can be used after previous sections without greater difficulty.

12. It is not too time consuming; the average time should be thirty to thirty-five minutes.

13. Postoperative convalescence is smoother, morbidity is less and the hospital stay is reduced.

14. Bladder complications are absent.
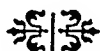
When removal of the uterus is indicated, as for fibroids and other non-malignant conditions, E. W. Munnell, Meigs and many other gynecologists believe that surgeons and gynecologists should be qualified to perform total hysterectomies. He admits that this process is followed by a slightly higher operative mortality and one must be most careful not to injure the ureters. Nevertheless, in skilled hands he believes hysterectomy should be performed to avoid possible malignancy of the remaining cervix at some later date. Also, the female's sexual response is unaffected by removal of the cervix. In fact, removal of the ovaries need not affect libido. For all these reasons it is urged that all future operators become skilled in performing total, rather than subtotal, hysterectomies. (*Richard A. Leonardo, M. D.*)

MODERN MANAGEMENT OF MEGACOLON*

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OUR purpose in this paper is to present a concise survey of the various methods of treating megacolon which are employed today, the results obtained from each and the complications encountered. Pathology, diagnosis and etiology will be discussed in so far as they affect treatment. We believe that a study which presents the best and latest thought on the subject should prove of value to the general practitioner, the pediatrician and the surgeon who may be called upon to treat this condition.

In view of the fact that so-called congenital megacolon is encountered only once in every 8,000[28] or 9,000[5] hospital admissions or once in 10,000 autopsies,[7] it is not surprising that we are today little closer to an explanation of this phenomenon than was Finney[8] when he wrote in 1908, "It is highly probable that more than one etiological factor is concerned in its production, as no single cause that has thus far been discovered will satisfactorily explain every case." His paper still stands as a classic and is well worth the attention of anyone interested in the subject.

Congenital idiopathic megacolon is rarely seen in an adult and for that reason the following case is reported:

CASE REPORT

A. M., No. 145023, a white woman, aged fifty-three, was admitted to the New York City Hospital on June 28, 1946, complaining of severe abdominal distention of forty-eight hours' duration. For as long as she could remember this patient had been subject to attacks of marked constipation and more or less abdominal distention. During the past ten or fifteen years she had been in the habit of taking two alophen pills twice daily for several days each week to relieve constipation and gaseous distention. Two years ago she suffered an acute attack of obstipation, abdominal pain and great distention. She had to take two whole bottles of magnesium citrate before she could secure relief at that time. There was no history of diarrhea, bloody or tarry stools. Her appetite, which was usually good, had fallen off lately and she had lost a considerable but unknown amount of weight.

The present attack resembled the others in that its onset was insidious, she could not evacuate her bowels and she observed a progressive enlargement of her abdomen. But her symptoms were more acute this time, with marked nausea, anorexia and dyspnea. She became frightened and for the first time sought medical advice.

Except as mentioned, her past medical history was negative. She had always been in good health and able to perform her duties as housewife and mother.

Physical examination on admission disclosed an acutely ill, poorly nourished and very apprehensive white female of fifty-three. Her temperature was 98.6°F., pulse 100, respiration 24 and blood pressure 150/90. The abdomen was markedly distended and tympanitic. Gentle palpation revealed no masses, spasm or tenderness, although the slightest pressure increased her general discomfort. The liver and spleen could not be felt. Rectal examination revealed a normal sphincter, an empty ampulla and no local obstruction. The remainder of the examination was negative. The blood count and urinalysis on admission were normal. A flat x-ray of the abdomen showed tremendous dilatation of the whole colon with gas and feces from the cecum to the lower sigmoid region where the distention abruptly ceased.

Efforts were made to decompress the bowel by intubation and to restore the fluid-electrolyte balance with infusions. The distention,
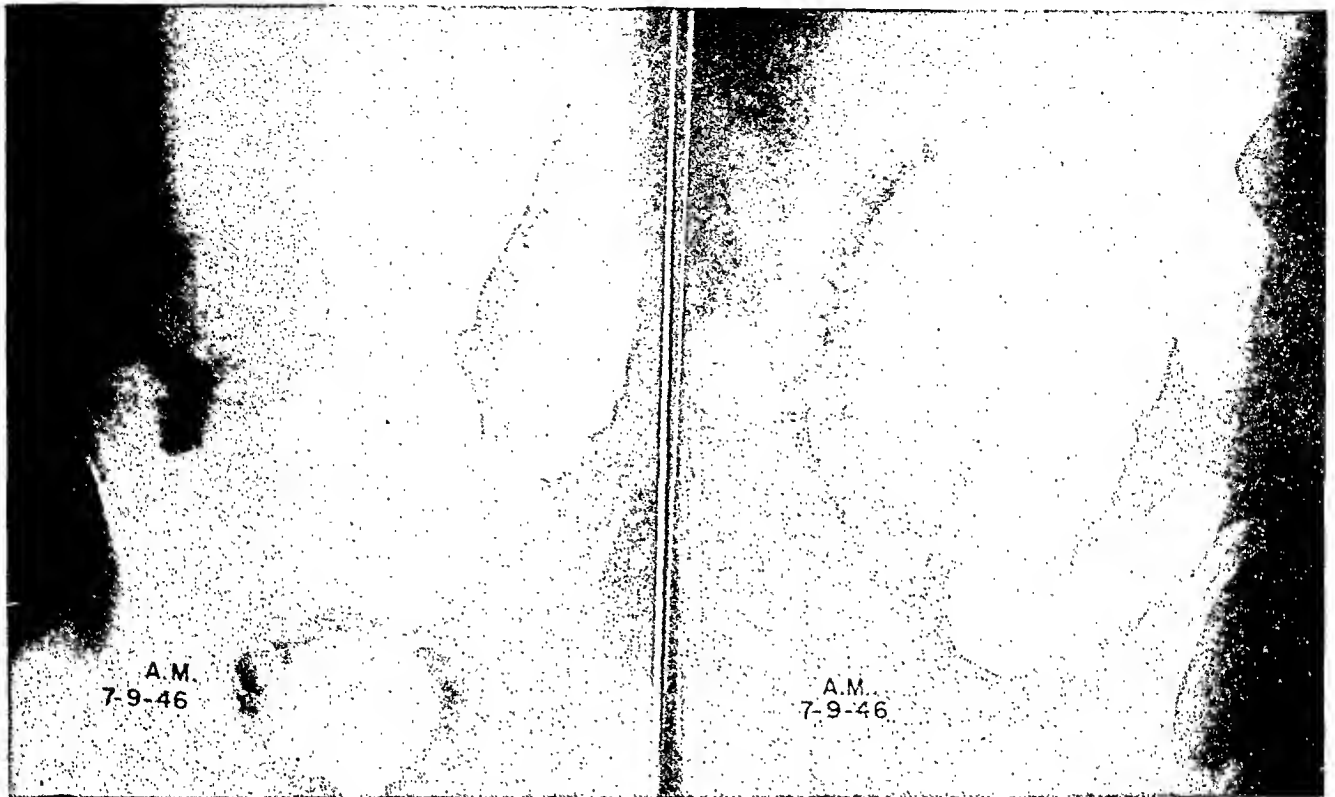
Fig. 1. Dilated, redundant sigmoid, ballooned out of pelvis, with obstruction due to volvulus at rectosigmoid.

however, was unrelieved. Further investigation of the distal colon by sigmoidoscopy and barium enema was considered too hazardous an undertaking in this acutely ill patient. Instead, with the patient prepared for operation, a spinal anesthetic was administered about twelve hours after admission. As no relief was afforded by this therapeutic test, an exploratory laparotomy was performed forthwith through a lower left paramedian incision for intestinal obstruction. The whole colon was tremendously distended with gas as far as the rectosigmoid. Distention was most marked in the descending colon and sigmoid which were enlarged to four times normal size. In this region the bowel wall was paper thin. The distal small intestine was dilated to a lesser degree. The descending and sigmoid mesocolon was extremely long and redundant. Due to the resultant abnormal mobility of this portion of the colon, an obstructing volvulus had occurred at the rectosigmoid junction. Manipulation of the bowel with reduction of the volvulus resulted in the sudden passage of a copious liquid stool with much gas. Marked immediate relief of the distention was observed. In view of the patient's poor condition no further surgery was attempted and the abdomen was closed.

The patient did well for a time after operation but the distention and obstipation re-curred and persisted despite paravertebral blocks and spinal anesthesia. Her course for several weeks was marked with a septic type of temperature in spite of supportive measures which included infusions, transfusions and intubation. A barium enema (Fig. 1) confirmed recurrence of obstruction at the rectosigmoid. On August 8, 1946, under continuous spinal anesthesia (which incidentally, did not relieve the distention) 40 cm. of dilated atonic descending colon and sigmoid were resected by the Paul-Mikulicz method. The proximal clamp was removed at seventy-two hours but when the distal clamp dropped off at ten days it was found that the distal loop had retracted and closed over to such an extent that the spur could not be isolated.

The patient was therefore returned to the operating room, the distal loop was freed up and clamps were applied to the spur. In ten days these cut through and the patient began passing small stools per rectum. Her general condition meanwhile showed marked improvement. As the colostomy showed no signs of closing at the end of six weeks, operative closure was effected on October 15, 1946. The postoperative course was uneventful. On the fifth day normal bowel function was reestablished and the patient was discharged, free of symptoms, twelve days later.

Fig. 2. Hemorrhagic ulcerated mucosa of resected sigmoid.

Fig. 3. Normal ganglion cells of Auerbach's plexus in wall of resected sigmoid. .

The report of the pathologist (James R. Lisa, M.D.) was as follows:

"*Specimen No. 29447—Gross Appearance:* The sigmoid measures 40 cm. in length and 14 cm. in circumference. It has its mesentery attached. The serosa is discolored, hemorrhagic, cyanotic and granular, especially along the antimesenteric border. When opened, the specimen is found to be filled with blood. The wall is thick and oedematous and the inner surface is studded with innumerable petechial hemorrhages. In only limited areas is the mucosa normal. (Fig. 2.)

"*Microscopy:* The wall is greatly thickened and oedematous. It has a myxomatous appearance and is diffusely infiltrated with lymphoid and plasma cells. Beneath the serosa are groups of foreign body giant cells, forming granulomata. The vessels are engorged and there are numerous fresh hemorrhages in the subserosa. The mucosa is extensively eroded and its surface is covered with well vascularized granulation tissue. Ganglion cells of the plexus of Auerbach are present and appear normal in both the involved and the uninvolved portions of colon (Fig. 3), although in the involved area there is oedema of the plexus and infiltration with eosinophiles and polymorphonuclears."

On January 28, 1947, six months after admission to the hospital, the patient was seen at follow-up examination. She stated that she had never felt so well in her life, that she had gained 10 pounds and was having three or four soft-formed stools daily. She was taking half an ounce of mineral oil daily but had taken no cathartics or enemas. There was no evidence of any recurrence on physical examination. The abdomen was flat and soft with a well healed left rectus scar and no hernia. Rectal examination was negative. A barium enema the same day showed no evidence of obstruction or recurrence in the sigmoid but there was persisting redundance and enlargement of the remaining large intestine. (Fig. 4.)

PATHOLOGY

Before attempting to discuss the etiologic factors of megacolon it will be useful to consider briefly the basic pathologic anatomy involved. As its name implies, this is characteristically a disease of the colon. The small intestines are seldom implicated. Very rarely a portion of the terminal ileum may share in an extension of massive colon dilatation but that is all. According to many investigators distention of the rectum is unusual, but Grim-

Fig. 4. Six months after operation; essentially normal sigmoid and descending colon; persistence of some redundance and enlargement of the remaining colon.

son[10] found it in half of his twenty-four cases. The whole large bowel from cecum to rectum may be involved (15 per cent of cases—Finney) but the sigmoid is the most frequent seat of the disease, being included in all or practically all cases.[8,10,18,21,24,33] Pathologic changes are limited to the sigmoid exclusively in more than one-third of cases.[2,8]

Dilatation is usually great and often tremendous. Bowel capacities of 40 pounds,[9] 16 liters,[23] 5 liters[30] and 4 gallons[27] have been reported. Hawkins[11] recorded a maximum circumference of 120.49 cm. and in Bach's case[1] the distended sigmoid measured 82.4 cm. in circumference and 10.5 inches in diameter. The transition from normal to dilated colon is usually gradual while that from dilated to normal bowel is most frequently abrupt. The lumen is sometimes distended with quantities of putty-like material having a sickeningly offensive odor, plus variable amounts of liquid feces and gas. Not infrequently large, stony-hard fecal concretions, sometimes as

large as a child's head,[30] have been found impacted in the bowel.

The colon wall in a typical case is greatly hypertrophied, being several times its usual thickness, and it has aptly been compared with a wet leather glove in appearance.[33] On the other hand an occasional case, such as ours, at operation shows an attenuated paper-thin colon, stretched to the bursting point,[4,5] in place of the usual hypertrophy. Even in such cases, however, the fixed specimen may show a thick apparently hypertrophied wall. When hypertrophy is present, it is seen in all layers including the mucosa which is pigmented and often ulcerated.

In practically every case the diseased colon is elongated and redundant as well as hypertrophied. The mesocolon also partakes of this abnormality. It is much enlarged, thickened and gorged with swollen blood vessels, lymphatics and nodes. Rarely is it shortened.[18]

Histopathology confirms the changes seen in the gross specimen. With few ex-

ceptions there is extreme hypertrophy of all the colon layers and nothing else. Rarely, as in the present case, thickening of the wall proves to be pseudohypertrophy, a myxomatous condition produced by edema and round cell infiltration. Sometimes the mucosal surface is ulcerated. Few reports have been found in which examination was directed specifically to the plexuses of Auerbach and Meissner. Finney[8] stated that these ganglia were examined and found normal in two cases while Tiffin[32] reported complete absence of Auerbach's ganglion cells in 5 to 7 cm. of normal upper sigmoid just distal to a grossly dilated colon in a twenty-one-month male child. In our case the plexus of Auerbach was normal in the uninvolved portion of the resected colon; in the diseased areas, however, it was edematous and infiltrated with polymorphonuclears and eosinophiles but the ganglion cells were apparently normal. (Fig. 3.) It is unfortunate that this investigation has been carried out so seldom in the past as it might throw some light on the etiology of the disease.

DIAGNOSIS

The clinical picture is essentially one of obstipation and meteorism. Although seen most frequently in the very young (70 per cent in the first few weeks of life—Finney) it may become manifest at any age. It is well established that megacolon in some cases is compatible with comparatively good health and may go unrecognized for several decades. It occurs predominantly in males.[5] Typically there has been severe constipation either from birth or over a long period of years. There may on occasion be alternate episodes of diarrhea which give no relief and fail to empty the bowel. Spontaneous stools are the exception. Weeks may pass without an evacuation and undigested food may then be noticed in the stool. There is progressive abdominal enlargement which remains unrelieved by the infrequent stools. Weight loss is progressive and may be extreme. Not infrequently the outline of the dis-tended colon can be traced through the thinned-out abdominal wall and vigorous peristaltic contractions can be seen. Whitehouse[33] mentions the doughy feel of the abdomen and the putty-like fecal masses which can occasionally be palpated. Respiratory embarrassment from pressure of the enormously distended gut is not uncommon.

ETIOLOGY

Hirschsprung[13] divided megacolon cases into groups according to age: those occurring in infancy which he called true megacolon, and those occurring in adult life which he considered pseudomegacolon. Age, however, is not a reliable criterion, for colonic dilatation due to organic obstruction occurs in infants and also megacolon which is, so far as we can tell, congenital and idiopathic, occasionally presents itself in an adult.

Enlargement and hypertrophy of the large bowel, at any age, secondary to recognizable organic obstructive factors such as tumours, bands, adhesions, stenosis, stricture and atresia constitute a large group of cases sometimes loosely designated "megacolon." Often cases due to organic obstruction are included in the same report with others for which no cause can be found. Indeed, many authors list obstruction as one of the main factors in the production of megacolon. This practice has been responsible for considerable confusion of thought in the literature and should, in our opinion, be abandoned. We believe very strongly that the term megacolon should be reserved for cases which are congenital and of primarily unknown origin even though certain of them, such as ours, may exhibit varying degrees of obstruction as a secondarily complicating factor.

Over two-thirds of the cases diagnosed as congenital idiopathic megacolon are infants, children and young people who have been severely constipated since birth, who go for days or weeks without bowel motion and in whom meteorism develops.

Such were the cases described by Hirschsprung[13] before the Berlin Congress for Children's Diseases in 1886.

A very few cases have been reported[27,30] in which the condition first became apparent well along in adult life, without any recognizable obstructive factor. It has been conjectured[28] that extrinsic metabolic disturbances such as avitaminosis, malnutrition, et cetera, are to blame for such deficiencies are commonly noted in these patients. It seems logical to us, however, to regard the deranged metabolism as effect rather than cause. We believe that these were probably mild cases of congenital idiopathic megacolon which for one reason or another escaped attention for years.

Other cases, likewise few in number, present an obstruction which is termed "functional" (dolichocolon) although it is really mechanical (kinking, volvulus). However, the primary disorder (elongation and hypertrophy) which makes mechanical obstruction possible is presumably related to embryonic development[25,27] and, therefore, congenital and idiopathic. Such factors are clearly seen in the patient we have presented. These are the cases that go unrecognized for years, with apparent good health and a few minor symptoms, only to develop suddenly into the classical picture of megacolon plus obstruction. Ligat[17] reported twelve which suffered from recurrent volvulus, all but one in adults. He stressed the importance of anatomical factors, elongated hypertrophied bowel and mesocolon. Eiss,[6] River[26] and Martin[20] have each reported a case of megacolon in an adult with obstruction due to volvulus or kinking, while Scott and Serenati[28] described six cases which they explained on this basis. The occurrence of obstruction is definitely a secondary complication and does not remove these cases from the congenital idiopathic category.

Detailed consideration of the numerous etiologic theories that have been advanced will be found in standard textbooks.[3,15,25]

It will suffice here to mention only those few which have received most attention in the past. One explanation is that a congenital defect in colon growth results in giantism of the large bowel either before or shortly after birth. Congenital defects, however, are frequently multiple while megacolon occurs almost universally in an otherwise perfectly normal child. Achalasia of the rectal sphincter has also been blamed, but many megacolon patients have normal sphincters, mechanical dilation of the rectum often fails to produce marked improvement and spasm of the sphincter is known to occur without producing megacolon. Some authors[12] believe that lack of segmental neuromuscular coordination is responsible but no explanation for the incoordination is offered.

Due to the fact that varying amounts of improvement and relief have followed treatment directed to diverse portions of the autonomic nervous system, there has been a tendency to ascribe megacolon to a neurogenic dysfunction or imbalance. If, indeed, imbalance is the responsible factor, there is as yet no clear understanding of what produces it or exactly how it works. Interruption of the sympathetic pathway (by spinal anesthesia or sympathectomy) is found useful in some cases but not in others. The same holds true of parasympathetic stimulants (mecholyl, prostigmin, physostigmin) and parasympathetic paralyzants (atropin, syntropan).

Obviously no one common etiologic factor which will explain every case has yet been discovered. Until more light is thrown upon this perplexing problem, therefore, we are forced to accept the theory of a mixed pathogenesis.[8,25] Megacolon is a syndrome rather than a disease. As such it is possible that it may be produced by any of the factors mentioned, by a combination of them or by a factor or factors as yet unknown.

TREATMENT

In view of the high mortality (70 to 80 per cent) in untreated congenital

idiopathic megacolon, it is obvious that some attempt must be made to ameliorate symptoms and to improve, if not cure, the underlying condition. Unfortunately, there is today a wide divergence of opinion as to how this can best be accomplished.

Conservative Management. All authors are agreed that, except in those advanced cases demanding heroic emergency measures, an intensive medical regimen should first be given a thorough trial. Such management includes a low roughage diet, rectal dilatations, colonic flushes, mineral oil, vitamins, mild catharsis and meticulous intelligent supervision over a considerable period of time in an attempt to establish a normal bowel habit. In addition to these measures, drug therapy[24] is recommended in the form of parasympathetic stimulants, such as mecholyl bromide[16] and eserin[5] or paralyzants such as syntropan.[14] But there are those[15] who believe that parasympathetic stimulants have a very limited usefulness and one case of death due to drug idiosyncrasy has been reported.[16] Mild cases may respond and be carried along fairly well on this treatment. However, in most instances medical therapy, with or without drugs, will have to be continued indefinitely and immediate success must not be expected even when drugs are used.

Spinal Anesthesia. This has been employed as a therapeutic adjuvant to the medical regimen,[29,31] with beneficial results in some cases. It is also recommended as a diagnostic test[28] of use in predetermining the value of sympathectomy.

Sympathectomy. Left lumbar sympathectomy has been credited with producing striking and lasting improvement in many cases which have proven refractory to medical measures. The operative mortality is low, 2.5 per cent in one series of 117 cases collected from the literature,[22] and many do well on a carefully supervised medical regimen after operation.[12] A note of warning should be sounded, however. If the newer drugs or spinal anesthesia fail to benefit the patient, there is little prospect of improvement from sympathectomy.

There is grave danger in males that sympathectomy will cause sterility (through lack of ejaculation) if performed bilaterally. In addition, bilateral sympathectomy, by interrupting pain pathways, blocks nature's warning of obstruction or impaction. Resultant perforation with peritonitis and death have been reported.[10] X-ray studies following sympathectomy consistently demonstrate that, while the condition of the large bowel may be somewhat improved, it never becomes normal, hypertrophy persists and there is always the danger of kinking, volvulus or impaction. Sympathectomy sometimes fails[10,24,33] even when done bilaterally.[32]

Ladd and Gross[15] recommend sympathectomy only in the early or moderately advanced case in which organic changes in the bowel wall are not too great. Of their six cases subjected to sympathectomy four had definite amelioration of symptoms which made further medical management much easier but two showed no significant improvement. Penick,[24] in a recent review of eleven patients (aged eighteen months to fifty-two years) upon whom left lumbar sympathectomy was performed, reported that seven had highly satisfactory results, three were greatly improved, although they still required cathartics or enemas, and only one was a complete failure. Nine of these were followed from one to eleven years. Penick recommends sympathectomy for any patient over two and a half years of age who has failed to improve under medical management but who has shown good response to spinal anesthesia or drug therapy.

In Grimson's series of twenty-four cases[10] four were subjected to sympathectomy. As a result, medical management was made easier temporarily in two and permanently in one. However, one patient died two months after operation, another had to be resected at one year because of serious impaction and a third died three years after sympathectomy of acute impaction with perforation. Rankin[25] believes that sympathectomy is the safest and surest surgical

measure for the relief of this pathologic condition. He mentions ten cases in which either bilateral lumbar sympathectomy or division of the presacral and inferior mesenteric nerves was done, with good results.

Colonic Resection. A. Subtotal resection: Certain authors[8,28,33,34,35] advocate subtotal resection of the colon in place of sympathectomy as the primary treatment of choice once the diagnosis has been established and adequate medical therapy has failed. They believe that while the operative mortality of sympathectomy is low, the operation does not appreciably alter the underlying disorder and therefore the risk of subsequent serious complications greatly outweighs the amelioration of symptoms that may be obtained.

The operative mortality of subtotal resection forty years ago ran as high as 48 per cent. Yet many then believed that the lasting benefits derived from it justified the risk involved. With the great advances that have since been made in preoperative and postoperative care, better understanding of fluid, protein and electrolyte requirements, recognition of the value of plasma and whole blood, the use of intubation for decompression and the introduction of antibiotics, chemotherapeutic agents and vitamins, the mortality rate has dropped until today it compares not unfavorably with that of sympathectomy.

In 1907 Loewenstein[19] reported 34 per cent good results in forty-four subtotally resected cases he had collected from the literature, with a mortality of 48 per cent. From 1909 to 1941 twenty-nine patients were subjected to subtotal resection at the Mayo Clinic[33] with satisfactory results in the sixteen who were followed and an operative mortality of 24 per cent. This was an overall report and does not fairly reflect improved figures for recent years. Ladd and Gross[15] reported ten subtotal resections with results as follows: five were greatly improved when last seen from one to twelve years after operation; one was

greatly improved for fourteen years and then suffered a recurrence; one was improved for eight months but died of other causes. One showed slight improvement, one none at all and one died of peritonitis following operation, making an operative mortality of 10 per cent in this small series. Yeazell and Bell[34] described six children in whom a two-stage subtotal resection was done with no deaths. Five of their cases had satisfactory results.

In those few megacolon cases complicated by a secondary obstructive factor (dolichocolon) subtotal colonic resection is generally recognized as the treatment of choice although an initial cecostomy or colostomy may be required for preliminary decompression. We believe that total resection is rarely indicated as a primary procedure in these cases.

B. Radical (total) resection: Megacolon may reappear in another segment of colon years after a subtotal resection.[5,10,15,28] For this reason Grimson[10] urges radical rather than subtotal colectomy at the primary operation in advanced cases. In 1945 he reported three successive cases of radical one-stage resection with ileosigmoidostomy. Good results were obtained in all three patients up to seven, eight and twelve months, respectively, when they were last seen. Ladd and Gross,[15] on the other hand, abandoned radical colectomy after losing three patients, two from leakage at the anastomosis and one from a perforating ulcer of the sigmoid stump one year after operation. It is possible that with further improvements in surgical care and supportive treatment, radical resection may come to be more widely accepted in resistant cases. A high degree of technical skill is of course required, but the outcome in an individual case depends at least to an equal extent on the physical condition of the patient. These patients are, at best, poor risks for radical surgery and much more experience with this method is needed before it can safely be recommended for general use.

SUMMARY

The pathology, diagnosis and etiology of megacolon have been reviewed. Methods of treatment advocated by various investigators have been presented and their results analyzed. A case of megacolon in an adult has been reported.

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VARIOUS ANESTHETICS IN ORTHOPEDIC SURGERY

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THE anesthesiologist of today has a wide variety of anesthetic agents to consider in providing anesthesia for orthopedic procedures. Final selection is influenced to a marked degree by several factors. Most important of these are: first, the temperament and physical makeup of the patient; second, particular likes of the surgeon; third, the skill, ability and experience of the anesthesiologist and fourth, the particular customs of the operating room. The skill, ability, and experience of the anesthesiologist is by far the factor of greatest importance in evaluating the usefulness of various anesthetic agents. In order to utilize our present day knowledge of the various agents it is extremely important that an anesthesiologist first evaluate the patient before final decision is made as to type of agent to be employed. This evaluation is most satisfactorily accomplished by a preoperative visit to the patient, and usually with a few questions and a brief conversation an expedient method of anesthesia may be selected.

Adequate medication preceding anesthesia is desirable and important. Frequently orthopedic procedures are carried out in multiple stages so that it is all the more desirable that these patients arrive in the operating room in a tranquil frame of mind. A wise rule to follow is that all patients should receive the benefit of some type of pre-anesthetic sedation unless there is some particular contraindication. Exceptions to this rule are usually made at the extremes of life. Premedication usually consists of pentobarbital sodium gr., $1\frac{1}{2}$ to 3 (0.1 Gm. to 0.2 Gm.) administered the night before operation and pentobarbital sodium, $1\frac{1}{2}$ gr. (0.1 Gm.) one and one-half hours before the stated time of operation. This should be followed by doses of morphine gr., $\frac{1}{8}$ to $\frac{1}{4}$ (0.008 Gm. to 0.016 Gm.) and scopolamine or atropine gr., $\frac{1}{200}$ to $\frac{1}{100}$ (0.00032 Gm. to .00064 Gm.) approximately one hour before the operation. The amount of pre-anesthetic sedation should be based entirely on information gained at the preoperative visit to the patient. Dosage of premedication should necessarily vary according to type of agent to be employed.

Morphine and Scopolamine. The production of analgesia with fractional doses of morphine and scopolamine[13] has proven very satisfactory in certain procedures such as application of a body cast or hip spica when little or no manipulation is required. If the desired analgesic effect has not been reached when the patient arrives in surgery, an additional dose of morphine and scopolamine may be administered intravenously. Employment of morphine and scopolamine analgesia facilitates many of the major plaster procedures for which complete anesthesia is not indicated.

Vinethene or Divinyl Ether. Vinethene has a limited field of usefulness in orthopedic surgery but it is valuable when a short-acting anesthetic is indicated and when profound relaxation is not required. When vinethene is employed, it must be borne in mind that the usual eye signs of anesthesia are not likely to be exhibited and that excellent analgesia usually is present when blinking of the patient's eyes is present or when there is evidence of widening of the palpebral fissures. It is in this stage of analgesia that many short manipulations of the wrist or ankle may be advantageously performed. Recovery from vinethene is prompt and is not usually attended by nausea and vomiting. This is an important factor in anesthetizing ambulatory patients since the rapid elimination of this agent permits almost immediate ambulation. Goldsmith[4] and associates and

Orth[11] and associates have demonstrated liver damage in dogs in administrations of vinethene lasting more than one hour. Martin and Rovenstine[10] have recommended that it should not be used to produce profound anesthesia or for operations requiring more than a few minutes to complete. It is our practice not to administer vinethene for procedures requiring more than ten minutes of anesthesia.

Nitrous Oxide. Nitrous oxide may be employed for reduction of certain fractures and dislocations, preferably when heavy pre-anesthetic sedation has been employed. Clement[1] uses nitrous oxide and oxygen alone for some orthopedic procedures, but for general usefulness many anesthetists combine nitrous oxide with other anesthetic agents. For orthopedic procedures nitrous oxide and oxygen combined with pentothal as described by Lundy[3] is daily proving valuable. This is a particularly advantageous combination to use when fireproof anesthesia conditions are desirable. Nitrous oxide shares with chloroform the distinction of freedom from the explosion hazard which is in marked contrast to such agents as ethylene, cyclopropane and ether. The difficulty of obtaining muscular relaxation with nitrous oxide and oxygen alone has been recently circumvented by the use of curare as an adjunct to nitrous oxide. Curare and nitrous oxide[3] and oxygen may be conveniently employed for orthopedic anesthesia problems as well as other types of surgery requiring relaxation.

Ethylene. Ethylene is used in much the same manner as nitrous oxide but should not be employed when x-ray or electrical equipment is being used.

Cyclopropane. Since the introduction of cyclopropane in 1934 by Stiles, Neff, Rovenstein and Waters,[15] this agent has enjoyed considerable popularity by both surgeons and anesthetists. Cyclopropane is the most potent of the anesthetic gases and may be employed to advantage for a number of orthopedic procedures. It has been our experience that the satisfactoriness of cyclopropane anesthesia is considerably enhanced in the majority of orthopedic situations if small amounts of ethyl ether are added to the inhaled mixture. Addition of small quantities of ether minimizes the parasympathetic effect that cyclopropane exhibits which is more pronounced in some individuals than others. Use of cyclopropane preceded by pentothal for induction has also proven very satisfactory.

Ethyl Ether. This year 1946 marks the one hundredth anniversary of the use of ether which was introduced for surgical procedures by Morton[6] at the Massachusetts General Hospital in 1846. Ethyl ether rightfully continues to carry a large share of the burden of inhalation anesthesia for orthopedic procedures. The flexibility of this agent, together with its relatively wide margin of safety, renders it a valuable agent. Ether would be even of greater serviceableness if more time and skill were devoted to its administration. Induction of ether anesthesia may be readily accomplished with vinethene, nitrous oxide, cyclopropane, ethylene or pentothal. The controllability of ether is considerably enhanced when an endotracheal tube is inserted. This is particularly so in orthopedic operations requiring placing the patient in a prone position. For example, in performing cervical laminectomy, ether has proven extremely satisfactory when administered by the endotracheal method. The value of ether for babies and small children is well established and ether anesthesia continues to be popular for children who undergo orthopedic procedures. Use of basal doses of tribromethanol with amylene hydrate (avertin) has been valuable in children on whom it is necessary to perform repeated operations under ether anesthesia. Basal dosage of 70 to 90 mg. Kg. may be employed. Avertin should be avoided, however, in operations which may prove to be of a shocking nature or operations likely to be attended with considerable hemorrhage. The incidence of using avertin as a basal anesthetic in connection with various orthopedic procedures has

been markedly reduced in recent years for two reasons: first, the increased popularity of pentothal sodium for induction of anesthesia and second, the wider application of adequate premedication.

Pentothal Sodium. The utility of pentothal is now well established in respect to the rôle that pentothal now plays in traumatic and orthopedic procedures. This in part is due to the rapid, smooth induction it usually affords. In dealing with traumatic problems a quiet induction is not only desirable but necessary many times to avoid further trauma to shattered limbs.

Anesthesiologists are constantly seeking combinations of anesthetic drugs that will provide satisfactory anesthesia with minimum doses of drugs employed. Lundy,[7] in 1928, coined the term, "Balanced Anesthesia" to refer to combinations of drugs employed to provide anesthesia whereby minimum quantities of several drugs are used. The benefit when employing this philosophy of anesthesia lies in avoiding toxic or near toxic doses of single drugs when profound anesthesia is required. Since the introduction of pentothal in 1934 by Lundy,[9] this drug has adapted itself especially well to this modern conception of anesthesia. By employing proper premedicant drugs in combination with pentothal and one of the gaseous anesthetic agents, the benefits of combined anesthesia are utilized and yet a relative wide margin of safety is assured. A popular combination for orthopedic procedures is pentothal with a 50 per cent mixture of nitrous oxide and oxygen. This combination of agents assures adequate oxygen supply yet considerable analgesia is provided by the nitrous oxide in this mixture, overcoming to some extent the inability of pentothal to provide good surface or skin anesthesia.

Pentothal sodium administered intravenously provides adequate relaxation for reduction of fractures and dislocations and for manipulations of the back and extremities. It is a standing rule in our department of anesthesia that whenever pentothal is administered, the patient is connected to a gas machine to breathe oxygen or mixtures of oxygen and nitrous oxide. This single measure has definitely increased the safety of pentothal anesthesia in our hands.

Curare. Since Griffith[5] introduced curare in 1942 for clinical use in anesthesia, the scope of usefulness of this drug has gradually increased. Curare may be administered in 20 to 40 unit doses intramuscularly to robust individuals about to receive inhalation anesthesia for orthopedic procedures. Used in this manner curare materially facilitates the induction of inhalation anesthesia. By using curare by the intravenous route it is possible to provide profound muscular relaxation with an extremely light plane of nitrous oxide and cyclopropane. For some patients this is a distinct advantage and is a method to be employed when the indications are present.

Regional Anesthetic Drugs. Procaine, pontocaine, metycaine and nupercaine are the drugs most commonly employed for regional and spinal anesthesia for orthopedic procedures. The choice of drug must of necessity be influenced by the skill and experience of the individual about to employ the drug. Procaine rightfully continues to be the drug most commonly employed for regional methods either alone or in combination with vasoconstrictor drugs. Metycaine has proven valuable when employed in a 2 per cent solution for brachial plexus block. Emergency orthopedic surgery is frequently carried out in the upper extremity of patients who have recently eaten, and the use of metycaine for brachial plexus block is many times a convenient means of providing anesthesia of the upper extremity when other methods are contraindicated. Pontocaine in our experience comes nearest to providing an all purpose agent for spinal anesthesia for orthopedic procedures of the lower extremities. It is potent and long lasting enough for most ordinary orthopedic operations. When pontocaine is combined with dextrose as described by Sise,[14] an anesthetic of definite hyperbaric character-

istics is provided and the level of anesthesia may be limited by employing the gravity technic. Prickett, Gross and Cullen[12] have recently reported the value of vasoconstrictors in increasing the usefulness of spinal anesthesia by employing epinephrine in combination with procaine. Whitacre and Potter[17] have pointed out the value of using vasoconstrictors in combination with pontocaine and nupercaine when employed for spinal anesthesia. Nupercaine may be employed for spinal anesthesia for orthopedic procedures which are destined to be time-consuming.

Refrigeration. Crossman[2] and associates have demonstrated the value of reduced temperatures in producing anesthesia of the extremities. The use of shaved ice to produce anesthesia of the extremities is well tolerated in poor risk cases and is the anesthesia of choice in desperate and moribund patients who must undergo amputation.

SUMMARY

Various anesthetic agents employed in orthopedic procedures have been briefly discussed. The value and the trend toward using combinations of anesthetic agents utilizing the beneficial qualities of several drugs has been stressed. Too much emphasis should not be placed on the anesthetic agent itself because the experience, skill and ability of the administrator is a factor of far greater importance than the particular agent employed. Recently Waters[16] has aptly stated, "It is not the tools but the way tools are used," that is the important factor in anesthesiology.

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THE CRITICALLY BURNED CHILD

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THE critically burned child presents a somewhat distinct and, indeed, a more exacting problem than does the critically burned adult. An extensive burn affects a relatively larger proportion of the child's body surface, particularly when trunk and chest are involved, since the extremities have a comparatively smaller percentage of skin area. Moreover, the child lacks the intellectual maturity essential for cooperation with the physician in the long and rigid control required for ultimate recovery. Therefore, certain specific phases in the treatment of the child patient are at variance with the therapy recommended for the adult.[1]

TREATMENT OF SHOCK

The initial step in dealing with critical burns is, of course, to win the crucial battle for immediate survival. In the author's judgment this engagement is often lost during the first twenty-four hours because the physician underestimates the oncoming shock and fails to fortify the patient with necessary systemic support. In such cases it has been found that primary or secondary shock appears from six to eight hours after inception of the burn.

Massive doses of plasma are required during this initial period, supplemented with intravenous injections or hypodermoclysis of isotonic electrolyte solutions.

PREPARATION OF WOUNDS

After shock treatment the second step is wound dressing. Whatever débridement is done before the initial dressings are applied should be carried out quickly and gently. Débridement should be kept to the minimum. The areas may be lightly cleaned with soap and water, rinsed with a physiologic saline solution and dried.

First dressings for severe burns are designed to control infection, to supply pressure that will check capillary dilatation and fluid loss and to prevent inspection and handling. The child patient is more comfortable with a dressing made of an ointment containing either an animal or a mineral base. (A 2 per cent xero form ointment is recommended.) When the time comes to remove this type of dressing, pain and bleeding are minimized. After the first twenty-four hours the administration of plasma should be discontinued as it is no longer of appreciable advantage and, in the writer's judgment, has no further place in the treatment of burns. Whole blood then becomes the life saving mechanism.

The initial dressings are left intact from ten to fourteen days. When those dressings are removed, complete débridement is done. If the burned areas fail to show healthy granulation tissue, grease dressings similar to the initial dressings are applied. These are changed every three days until granulation tissue appears. If granulation tissue is present, an alternating change of dressings from grease to wet and from wet to grease is advisable while preparing the wounds for skin coverage. The wound bacteria are resistant to local treatment and an occasional change in type of dressing provides more definite bacteriocidal action. For the same reason a change of solution is recommended for wet dressings: Dakin's, boric acid, normal saline or mild acetic acid. When wet dressings are being changed, the layer of gauze next to the wound should be removed cautiously to limit the amount of bleeding. Before the change is made all fresh dressings should be prepared for immediate application. In order to replenish the supply of blood and to bring the

hemoglobin up to at least 80 per cent, it may be necessary to give the patient whole blood. At any rate the blood should be checked during the period of wet dressing changes. The change of wet dressings each day or the change of grease dressings at three-day intervals requires a bedside routine that is a factor of some significance in handling the patient.

The surgical team is properly masked and gowned, a sterile bed sheet is spread over the foot of the bed and on it are placed tongue depressors, a bowl, scissors and two or more heavy roller bandages. When grease dressings are employed, an assistant cuts as many strips of heavy roller gauze, 4 by 8 inches, as are needed. With the use of a tongue depressor, the ointment is removed from the container and spread on the gauze. One 5 yard heavy roller bandage is placed in the bowl containing saline solution. One assistant now removes the soiled dressings and the freshly anointed strips are immediately placed over the wound. Then wet heavy roller bandage is firmly applied. Following this, come the elastic roller bandages and adhesive tape. If the granulating areas are exuberant, it is good practice to incorporate marine sponges just over the greased dressings. Incorporating the sponges in the heavy roller bandages gives them the added pressure needed to help flatten the granulating bed.

So much has been said about preparation of the wounds because a critically burned child furnishes only limited donor areas and cannot afford to lose even a small percentage of the grafts.

HOMOGRAFTING

To wait for third degree burns to close over with scar tissue is not a realistic approach. Frequently, during the third to sixth week following inception of the burn, sloughing is not complete, the granulation area appears dull and somewhat exuberant, the protein loss is high, there is a negative nitrogen balance and the hemoglobin is below 70 per cent despite several whole

blood transfusions. At this period homografting may save the child's life. The third step then in the critically burned child's progress is homografting. Homografts will bridge the gap between the extreme toxic stage when autografting might prove fatal and the fourth step, which is autografting. In this mid-stage of treatment, homograft coverage seals the wounds and makes possible the attainment of a positive nitrogen balance, an increase in food intake and an improved blood picture. Naturally, autografting must be planned from the outset and the wounds must be treated with this purpose in mind.

Repeated homografting to await scar coverage is a false hope if the burned areas are extensive. When the grafts liquefy, only a small part of the burned areas may be flooded over with scar epithelium. Circular areas seldom close over. In the meantime the patient's morale is seriously affected. Gain in weight or even maintenance of body weight is extremely difficult; the nitrogen imbalance grows; debilitation becomes more pronounced and fatal termination threatens.

Experience indicates that a second coverage of homografting from the original donors fails to "take" and skin from new donors adheres for a lesser time than did the original donor take. For although the first sheets of skin may remain viable for from three weeks to two months, the second crop remains viable for no more than two weeks. It is a ticklish question as to how many times homografting should be repeated. In the author's experience, in the case later to be described, homografts were made twice.

Up to this point no mention has been made of the food intake of the critically burned child. From the beginning he should be placed on a high protein, high vitamin diet but frequently he will refuse food, nor will his resistance collapse under persuasion be it gentle or firm. He probably finds amino acids distasteful, in which event tube feeding is the sole recourse. Amino acids are to be prescribed as a daily supple-

ment to the regular food intake, 4 Gm. per Kg. Eggnogs, milk and cream and broths with a minimum amount of salt, to prevent edema, also should be given. Administration of multiple vitamin concentrates is suggested.

In preparation for the first homografts, supplemental feedings of amino acids are carried on for a week or ten days in advance of surgery. A thick mixture (1 ounce to each 20 pounds of body weight) is placed in a large ear syringe and forced into the feeding tube. The child should be placed in a semisitting position in order to prevent vomiting, as the amino acids are sent through the tube. No water is permitted for one-half hour before or after the feeding. Tube feeding is continued for two weeks after either homografting or autografting.

One week before grafting the grease dressings are discontinued and only wet dressings are employed. The dressings are wet every three hours day and night with daily changes of dressings. Saturation of the dressings is necessary to prevent extreme pain and free bleeding when they are removed next day. On the day of surgery the dressings are changed early in the morning. Postoperatively the wet dressings are continued for six days. Thereafter, between grafting stages ointment dressings are reemployed to add to the child's comfort. During the homografting process, 250 cc. of whole blood is administered postoperatively at the bedside; this is repeated on the third day.

The initial homografts are done as quickly as the granulating tissue is reasonably clean, usually three or four weeks after the burn. To obtain the greatest percentage of coverage it is advisable to suture the homografts under moderate tension. If a general anesthetic is contraindicated, then the homografts can be brought back to the original size and tension by glueing one side of the graft on gauze and stretching and glueing it down to the opposite side. Immediately afterward the graft with the attached gauze is placed on the wound.

The same procedure is followed with each homograft taken from the donors.

The covered wounds are then snubbed on with two or more heavy roller bandages followed by an elastic bandage and adhesive plaster. Two or three weeks later if the patient has gained weight, if the red cell count is 4,000,000 and if the hemoglobin exceeds 80 per cent, the patient can be prepared by daily changes of wet dressings for the first stage of autografting. As has been indicated an additional homograft or two may be necessary before the child is physically fit for the autografts.

AUTOGRAFTING

When the child is ready to begin the final stage, permanent coverage of his wounds, the routine of tube feedings, whole blood administration and the application of wet dressings, as previously described in the homografting process, must be repeated. Sometimes there is a problem in skin grafting as to the sequence of events: Should the graft be taken from the patient before or after the wound is prepared? When dealing with a contaminated area, such as a burn wound, it is a sound principle to start from the clean site and advance to the contaminated site; therefore, in all burn wounds the grafts should be taken first.

Because infection is ever present, penicillin is used routinely. During surgery the proportion of 100,000 units per 5 cc. of saline solution is injected beneath the grafts at several points with a fine needle. Streptomycin, 100 mg. per cc. of saline solution, is added when the culture shows Bacillus pyocyaneus.

The child with an extensive burn provides only limited areas from which grafts can be had for the coverage of wounds. With donor sites limited, it is necessary that the first graft be taken no thicker than 0.006 or 0.008 inch in order that a second and a third crop can be garnered later. Nor is it advisable to subject the child to repeated trips to surgery by removing only small amounts of skin at each

operation; instead, as many as six or eight drums of skin should be taken at one time.[2]

When after intervals of a month or so the second and the third grafts are taken from the same sites, the dermatome should be set at 0.008 inch as the epithelial layer is exceedingly thin and friable. The surgeon must resist the temptation to increase the thickness beyond 0.008 inch on the second and third drums as there is danger of picking up all of the subepithelial elements. To get the requisite thinness of 0.006 or 0.008 inch a dermatome is used. The brush employed to apply the cement to the drum and to the donor site is placed in ether until it is needed, at which time the ether is shaken off. The glue must be thin when applied to the donor site and drum and the surgeon must pause until the glue is entirely free from sheen. The cutting blade, which is held at calibrated distances from the drum, is tested with "shims" of 0.006 and 0.008 inch. The shim is a unit of measurement similar to the one used by automobile mechanics in testing distances of tappets.

The late Dr. Earl C. Padgett, when the dermatome was first manufactured, suggested to the author the idea of double checking with shims. This test will frequently reveal a difference of from 0.002 to 0.004 inch at either side of the starting point of the drum although to the naked eye both sides appear equal. Another such test can well be made at the halfway spot on the drum so that, if necessary, the calibration can be reduced one or two notches before proceeding to the end of the drum. This provision is particularly needed if grafts are to be taken from the inner aspects of the thigh and from the abdomen as the skin in these areas is much thinner than is supposed.

Removal of the graft from the drum is made with extreme caution as it will tear readily. Upon its removal the graft is placed over a granulating area and the epidermis is immediately covered with a sulfonamide powder to nullify the adhesiveness of the glue.

Small grafts are not favored; if placed unsutured, many of them slip away whereas to suture them, dangerously prolongs the child's surgical experience. If stamp grafts or other grafts are applied without normal tension, from 20 to 30 per cent of their potential is actually lost. If instead a graft 4 by 8 inches is cut, it can be stretched to 4 by 10 or 12 inches by means of a new type of massive autografting which will be described.

The graft is basted on one side while one of the surgical assistants stretches it under moderate tension; meantime the surgeon cuts large slits in the graft longitudinally or vertically to provide added width or length. By this method the area has been given the fullest amount of skin coverage and the take of the graft extends over a greater area. Flooding over by epithelium into the interstices occurs rapidly, within two weeks. As soon as the grafts are taken the donor area is sprayed with thrombin solution to limit blood loss at the donor site.

It has been estimated that following the removal of a split graft of approximately 30 square inches the donor site will lose as much as 50 cc. of blood. When four or five drums or more of split skin are removed at one operation, the amount of blood loss is a serious consideration. The child's susceptibility to shock, plus the blood loss from donor sites, necessitates the use of large amounts of whole blood beginning in the surgery and carried on for four days postoperatively. As much as 750 cc. may be necessary for the first twelve hours for a child four or five years of age. Donor sites will stop losing proteins in four days and will heal over with epithelium in six or seven days.

The following case report demonstrates application of the measures described on a child with very extensive burns.

CASE REPORT

M. S., a boy of four, suffered extensive third degree burns on May 27, 1946, when his flannel pajamas caught fire while he was playing with matches. (Figs. 1 and 2.)

1. Full drum of skin 4"x8" contracts to approx. 3¼"x7" when removed from drum.

2a. Normal skin tension obtained by drawing on Allis forceps.

2b. Slits are made in graft.

3. Granulating areas will be flooded with epithelium within 2 weeks.
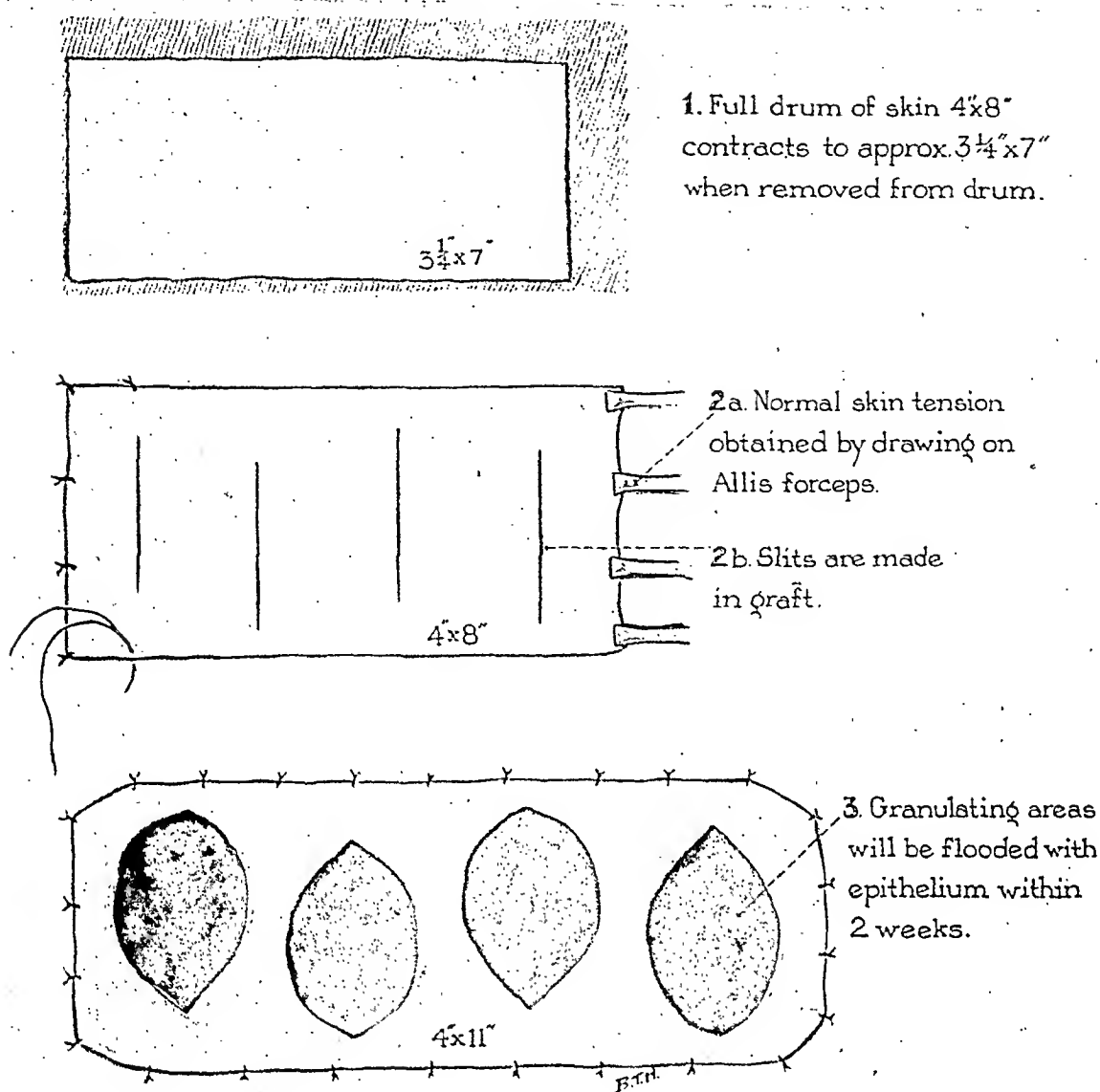
FIG. 1.

When first seen at Illinois Masonic Hospital, Chicago, six weeks after the burn, the affected areas showed numerous patches of necrotic skin and exuberant granulating areas. Removal of the dressings resulted in free bleeding. Following a two weeks' regimen of daily change of dressings (firm wet heavy roller bandage), of tube feeding with high protein concentrates and of several whole blood transfusions, the patient was taken to surgery.

Four full drums of thin split-thickness skin were taken from the mother and father and sutured under normal tension. Two dermotome drums of thin split-thickness skin were taken from the inner aspect of the patient's thighs to cover the right elbow and axillary region.

The second surgery was delayed eight weeks owing to an acute hepatitis and acute nephritis. The icteric index climbed to 80 but returned to normal under daily intravenous administration of 50 per cent glucose solution. At this surgery seven full dermatome drums of thin split-thickness homografts were snubbed on the wounds without an anesthetic.

The third surgery was six weeks later when a second crop was taken from the inner aspects of the patient's thighs and one dermatome drum from the outer aspect of the patient's thigh. After another six weeks' interval came the fourth surgery. Five dermatome drums of thin split-thickness skin were taken from the patient's abdomen, the gluteal regions and the outer thighs. Four weeks later the patient again submitted to surgery, at which time four dermatome drums of thin split-thickness skin were taken from his abdomen, the gluteal regions and the outer thigh.
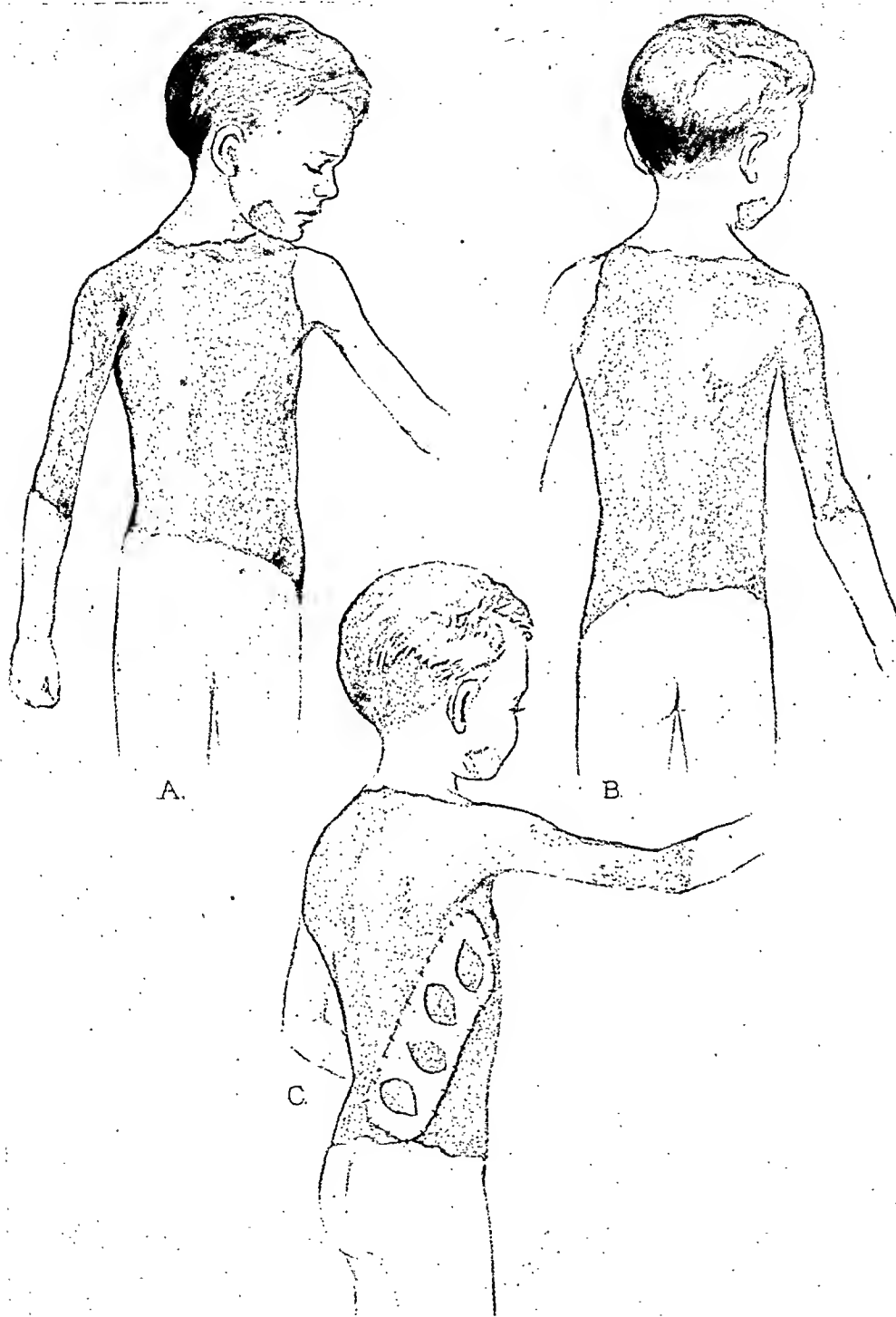
FIG. 2. A, third degree burn front; B, third degree burn back; C, full drum of skin 4 inches by 8 inches sutured to wound; slits made in graft.

Thus eleven full drums of homografts and fourteen drums of autografts, or a total of twenty-five drums of skin, resulted in complete coverage.

SUMMARY

In summary, the following points might be reviewed:

1. In the case of the critically burned child prepare to modify some of the technics recommended for adults.[1]

2. Anticipate primary or secondary shock from six to eight hours after inception of the burn. Give massive doses of plasma and electrolytes to correct circulatory imbalance.

3. Discontinue plasma after twenty-four hours and substitute whole blood.

4. As soon as granulation of wounds begins alternate the dressings between grease and wet.

5. If the patient is listless, is deficient in nitrogen and has a low hemoglobin, institute homografting preliminary to autografting.

6. Perform autografts with the thinnest possible thickness of skin (0.006 to 0.008 inch) in order to obtain rapid healing of donor sites so that removal of a second, third or fourth crop from the original donor sites can be done without too great an interval between surgical interventions.

7. Take massive autografts to cut down subsequent autografting. A new system of slitting large grafts gives increased coverage.

8. Organize teams for both taking grafts and applying grafts. A three-man team is needed for the application of the grafts. The patient should not be under surgery for more than one hour.

CONCLUSION

Homografting may become a life saving measure in the treatment of the critically burned child and represents a logical step in the preparation of the child for ultimate coverage of wounds with autografts.

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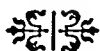
RICHTER'S HERNIA*

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RICHTER'S hernia is a form of strangulated hernia in which less than the full circumference of the bowel is caught by the hernial ring. (Fig. 1.) The or omentum, on account of its elasticity, acts as a cushion or buffer and this delays the onset of strangulation." Richter's
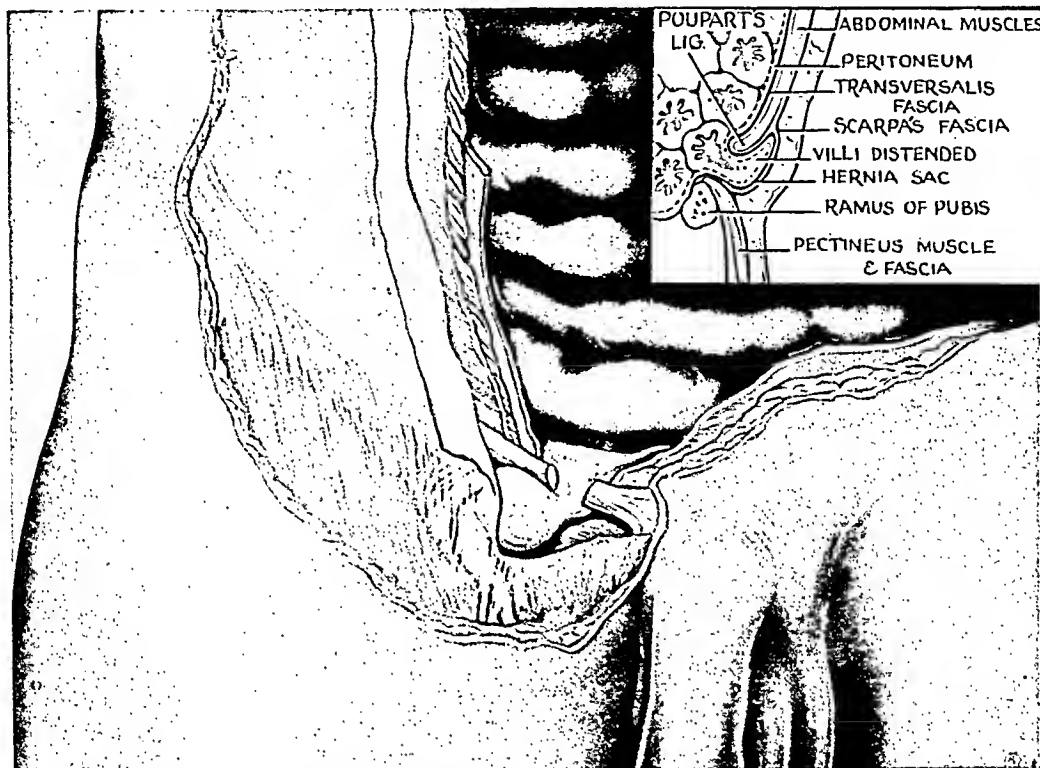
FIG. 1. *Anatomical Study*—Femoral Hernia: Poupart's ligament has been divided. Searpa's fascia shown blending with deep fascia of thigh 3–4 cm. below Poupart's ligament. This barrier completely covers the fossa ovalis thus directing the hernia upward.

terms "small hernia"[1] and "partial enterocele"[3] are found in the literature. Richter's original discussion,[1] probably first published in 1778, was the first to segregate this type of hernia although it is not clear that strangulation was considered necessarily a part of the picture. This variety of hernia is surgically urgent because gangrene occurs much earlier than in ordinary strangulations. Watson[13] states that "this is undoubtedly due to direct pressure exerted on the intestine by the constricting ring. In ordinary enterocele the mesentery hernia may be difficult to diagnose because of its small size, because the pain is often poorly localized and minor in character and because obstructive symptoms are extremely variable. The latter may vary from obstipation to diarrhea. Occasionally bowel movements are perfectly normal. Differential diagnosis of Richter's hernia through the femoral "empty space" is often difficult since it may not be distinguished from enlargement of the lymph nodes in this region. They are both deflected upward by the extension of Scarpa's fascia down the thigh which blends with the deep fascia

some 3 to 4 cm. below Poupart's ligament. (Fig. 1.)

The patients studied here were admitted to the Second (Cornell) Surgical Division between December, 1917 and July, 1945. We have included only those in whom the surgical disorder was definite and unmistakable.

ANALYSES OF PATIENTS

Age. The average age of these patients was sixty-one years. The hernias occurred most often in those in the sixth and seventh decades; (Fig. 2) the youngest patient was forty-one, the oldest eighty-nine. Fowler[3] states that the condition "appears to be limited to adults." Gouvernale et al.[9] state that the condition is seldom seen in children but mention Rhodes' case of a baby three weeks old. Patients eleven years of age and under were not admitted to our service but we noted a significant lack of young adults.

Sex. The exact ratio of females to males is not clearly stated in the literature. It is said[3] that hernias of this type are more common in females. Another author[9] goes so far as to say that females outnumber males by three, or two to one. In our series there were nine males and eight females. These figures are weighted by the fact that the Bellevue Hospital surgical population is predominantly male, there being two male wards to each female.

Site of Hernia. It is stated[1] that this type of hernia can occur at any body orifice. Treves,[2] citing fifty-three cases, noted thirty-eight femoral, thirteen inguinal and two of the ventral variety. The femoral variety makes up 90 per cent of all Richter's hernias according to Gouvernale.[9] In our series fifteen were of the femoral variety and two were inguinal. Both inguinal cases occurred in males.

Contents of Sac. The clinical size of the sac varies considerably depending on the amount of peritoneal fluid present and whether other structures, e.g., the omentum or appendix are present. It can be very small and in one of our patients Case

xiii the presence of a tiny inguinal mass was first discovered in the operating room during abdominal preparation. When muscles relax under anesthesia, the involved bowel frequently slips back into the abdominal cavity and in such a case it may be
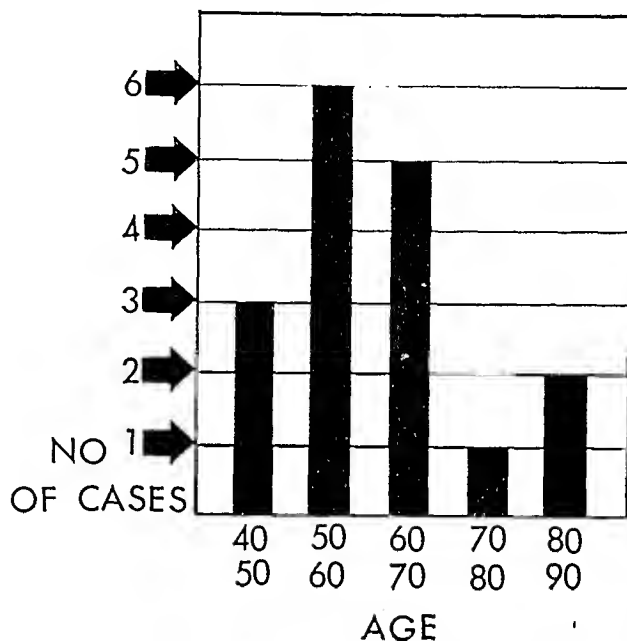
Fig. 2. Graph showing distribution of cases according to age.

impossible to retrieve or see the loop through the hernial opening. In such cases laparotomy is always performed. The ileum is the most common portion of the bowel involved[9] but the colon and jejunum are also found to be common sites of involvement. In all of our patients small bowel was found. In ten cases the bowel was viable and after a suitable period of observation was returned to the abdomen. Six patients showed gangrenous gut and one presented gangrene with perforation and the formation of a fecal fistula. Duration of obstructive symptoms in these seven patients with gangrene averaged just over nine days, the shortest was four and the longest was twenty-one days. When gangrene was absent, obstructive symptoms averaged 4.25 days; the shortest was of twelve hours' duration and the longest fourteen days. In two patients incarcerated omentum was found. The sac contained bloody fluid in eight and clear fluid in five patients. The presence of fluid was unrecorded in four cases.

TABLE I

SUMMARY OF CASES

Case No.	Hosp. No.	Date of Admission	Initials	Sex	Age	Previous Hernia	History	Side	Type	Contents of Sac	Operative Procedure	Choice of Anesthesia	Results
I	11-D2	12-1-17	A. F.	M	82	None	Abdominal pain, vomiting, obstipation for 2 weeks	L	Femoral	Omentum, and small bowel bloody fluid	Femoral incision, catgut repair	Local	Wound infection, bronchopneumonia, died 40th postoperative day, no autopsy
II	21-D2	5-8-21	H. Y.	M	53	Many yr.	Incarceration for 4 days; vomiting, obstipation for 2 days	L.	Femoral	Small bowel, bloody fluid	Femoral incision, catgut repair	General	Uneventful course; follow-up four months; no recurrence
III	23-D2	6-23-21	W. C.	M	41	5-6 yr.	Sudden effort resulting in incarceration; vomiting, obstipation for 12 hr.	R	Femoral	Small bowel, bloody fluid	Vertical incision crossing Poupart's, inspection of bowel in abdomen; Bassini and femoral repair, catgut	General	Uneventful course; discharged healed; no follow-up
IV	68-D2	2-17-23	B. K.	F	47	10 yr.	Abdominal pain, anorexia, nausea and vomiting for 1 day	R	Femoral	Small bowel, clear fluid	Femoral incision, catgut repair	Local	Uneventful course; no recurrence in two months
V	69-D2	5-21-24	J. S.	M	68	None	Mass appeared in inguinal region 3 weeks before admission; drained feces 1 week	R	Femoral	Fecal fistula from 1 inch defect in small bowel strangulated in femoral ring	Right lower split rectus incision small bowel reduced; defect repaired in 3 layers, abdomen drained	General	Sinuses on thigh healed two weeks after two segments of fishbone were discharged; no recurrence
VI	78-D2	11-5-26	J. K.	M	58	None	Inguinal pain on exertion 2 weeks, abdominal pain, vomiting, obstipation 3 days	R	Femoral	Gangrenous small bowel ¾ circumference	(1) Femoral incision extended on to abdomen, gangrenous bowel exteriorized; (2) gangrenous bowel excised end-to-end anastomosis with Murphy button	1. Local 2. General	Passed button twelfth day; wounds healed; recurrence upper end incision one year
VII	39-D2	2-2-28	J. P.	M	67	8 yr.	Abdominal pain with mass in right groin 1 day	R	Femoral	Small bowel with bloody fluid	Femoral and right lower rectus incisions; catgut repair of femoral defect	Spinal	Paralytic ileus and bilateral bronchopneumonia; died 3rd postoperative day
VIII	53-D2	1-26-34	K. B.	F	69	None	Abdominal pain, distention, vomiting and obstipation, 6 days	R	Femoral	Gangrenous small bowel ½ circumference	Femoral incision, gangrenous area excised; transverse repair; catgut repair of hernia	Spinal	Paralytic ileus and bronchopneumonia; died 1st postoperative day
IX	14163-35	3-25-35	L. O.	F	52	None	Abdominal pain, vomiting and obstipation, 6 days	L	Femoral	Small bowel, clear fluid	Femoral incision, catgut repair	General	Uneventful course; no recurrence in two years
X	43481-35	9-8-35	M. F.	F	59	1 yr.	Abdominal pain, vomiting and obstipation, 8 days	R	Femoral	Small bowel, clear fluid	Femoral incision, catgut repair	Spinal	Paralytic ileus; bronchopneumonia; died postoperatively 1st day
XI	2532-37	1-14-37	S. P.	M	70	20-30 yr.	Abdominal pain, vomiting and obstipation, 4 days	R	Femoral	Gangrenous small bowel ¾ circumference with bloody fluid	Femoral incision, bowel resected Murphy button anastomosis, abdomen drained	Local	Died 1st postoperative day; pulmonary edema

TABLE I (Continued)

Case No.	Hosp. No.	Date of Admission	Initials	Sex	Age	Previous Hernia	History	Side	Type	Contents of Sac	Operative Procedure	Choice of Anesthesia	Results
XII	4687-37	2-28-37	A. C.	F	58	None	Abdominal pain, vomiting and obstipation, 2 days	L	Femoral	Small bowel with clear fluid	Femoral incision, catgut repair	Local	Uneventful course; no recurrence 1½ year later
XIII	19221-43	4-21-43	R. B.	F	45	None	Abdominal pain, vomiting and obstipation, 5 days	R	Femoral	Gangrenous small bowel, bloody fluid	Right rectus incision; resection small bowel; end-to-end anastomosis	General	Paralytic ileus; bronchopneumonia; pulmonary edema; died first postoperative day
XIV	47515-44	10-18-44	A. H.	M	64	1 yr.	Abdominal pain, vomiting and obstipation, 2 days	L	Femoral	Small bowel, clear fluid	(1) Left rectus incision, hernia reduced; (2) femoral incision, catgut	1. General 2. Local	Uneventful course; no recurrence one year
XV	14387-45	3-21-45	W. E.	M	58	None	Abdominal pain, vomiting and obstipation, 4 days	R	Inguinal	Small bowel	Inguinal incision, reduction, Halstead repair with catgut	General	Uneventful course; recurrence in ten months
XVI	26922-45	6-5-45	A. S.	M	65	5-6 yr.	Incarceration of right inguinal hernia 1 week	R	Inguinal	Gangrenous small bowel and omentum, bloody fluid	Femoral incision, gangrenous bowel retracted into abdomen; incision drained, low midline incision. Gangrenous bowel resected end-to-end anastomosis	Spinal, General	Uneventful course; no recurrence in seven months
XVII	31990-45	7-5-45	L. S.	F	89	None	Abdominal pain, vomiting and obstipation, 1 week	L	Femoral	Gangrenous small bowel and omentum, bloody fluid	Femoral incision, resection small bowel, end-to-end anastomosis Murphy button	General	Uneventful course; passed button 15 days; discharged well
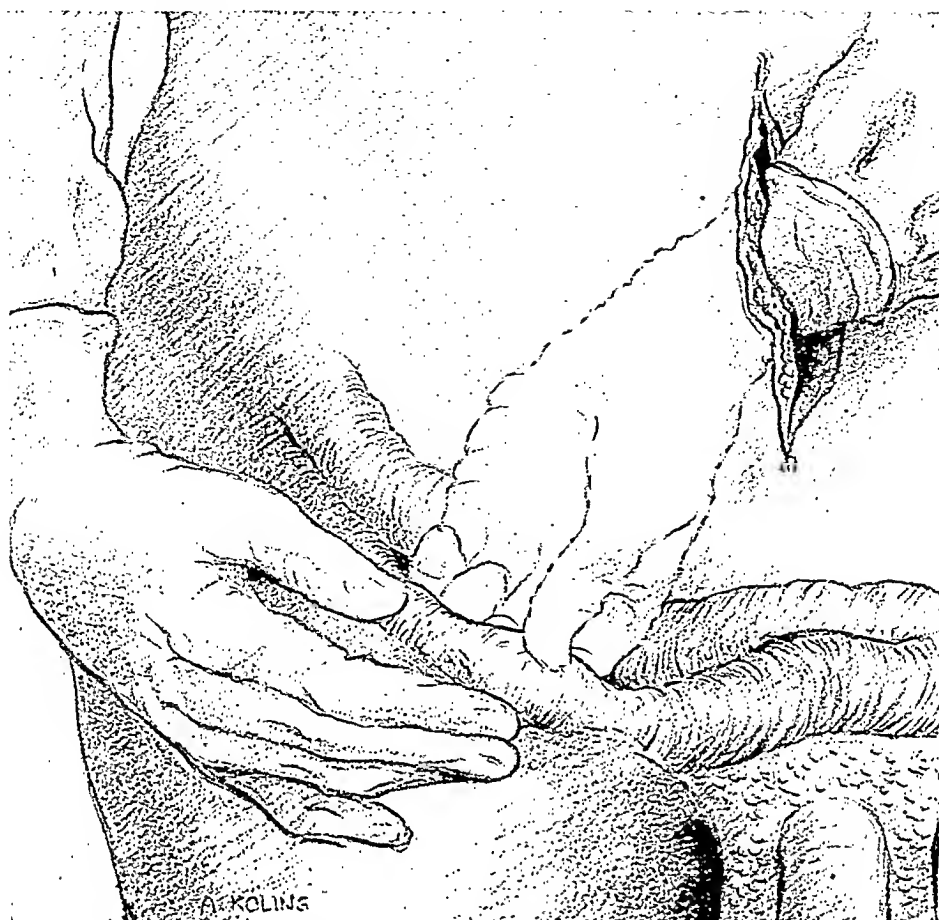
FIG. 3. Artist's sketch showing technic of bimanual reduction of hernia.

History of Previous Hernia. In nine cases the patient was unaware of any difficulty until the onset of acute symptoms. The remaining eight patients had vague histories of known hernias ranging from one to thirty years in length of time.

Treatment. The treatment is prompt operation, the earlier the better. However, if the patient's condition is poor due to obstructive symptoms, time is taken to restore fluid and electrolyte balance. Closed reduction is never attempted. No operative routine is suggested as each patient presents a different combination of problems. Only two things are of prime importance: (1) to reduce the hernia and (2) to be assured of viability of the strangulated bowel. Elegant hernia repair can wait until a later operation unless the patient's condition allows it.

A glance at the column entitled operative procedure in Table 1 reveals a num-

ber of ways of handling this problem. With minor variations they fall into three groups: (1) Those handled by an incision directly into the hernial sac, inspection of bowel, resection if necessary and hernial repair (ten cases). (2) Those handled by laparotomy at a distance from the hernia, reduction and inspection of bowel with resection if necessary (four cases). (3) Those handled by two incisions or by a vertical incision across Poupart's ligament (three cases).

The authors believe that the last method is to be avoided if possible. The making and repair of two incisions is time-consuming and frequently unnecessary. The need for a second incision arises for three reasons: (1) The bowel reduces and cannot be retrieved for inspection through the hernial opening or (2) the mesentery is too short to allow withdrawal of sufficient bowel for inspection or repair or (3) the

hernial opening is too small to allow adequate bowel to be withdrawn. In our experience a lower rectus incision followed by reduction is advisable in certain cases. A combination of pressure over the hernial mass and traction on the strangulated loop from within the abdomen is safely done. (Fig. 3.) The mesentery relaxes, blood supply is aided by hot packs and after fifteen minutes or so very doubtful bowel wall will begin to show peristalsis. If resection is necessary, it can be done under optimum conditions. The hernia may be repaired intra-abdominally as suggested by Cattell[5] or be left for routine elective repair later on.

If the hernia is clinically small and if gangrene is probable, then we believe this approach to be mandatory. The penalties following improper anastomosis done under difficulty or those following return of nonviable gut to the abdominal cavity remain enormous. The penalties of an accidental rupture of gangrenous bowel in the manipulative reduction have become progressively smaller due to advances in chemotherapy. If the hernia is large or recent and if one is reasonably sure gangrene is not present, routine reduction and herniorrhaphy should be attempted.

RESULTS

The mortality in our series was six patients in seventeen or a little better than one in three. It is interesting to note that three of these deaths occurred in patients in whom viable bowel was found. The mortality of patients requiring resection was 50 per cent. Four patients died with paralytic ileus and one died on the fortieth postoperative day following wound infec-

tion and bronchopneumonia: The remaining death ensued due to cardiac failure with pulmonary edema.

Our late follow-up is incomplete. Three patients never returned following discharge. The other eight were followed for a period of from two to twenty-four months. Recurrent hernias were discovered in two patients.

SUMMARY

Seventeen cases of Richter's hernia are presented. Anatomic considerations and methods of treatment are discussed.

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Case Reports

INTERMITTENT RAYNAUD'S PHENOMENON RESULTING FROM NON-UNITED FRACTURE OF THE NAVICULAR BONE

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ACCORDING to Allen, Barker and Hines,[1] "Raynaud's phenomenon may be defined as episodes of constriction of the small arteries and arterioles of the extremities resulting in intermittent changes in color of the skin of the extremities such as pallor, cyanosis or both. Raynaud's phenomenon may occur primarily as in Raynaud's disease or it can occur secondarily in association with a number of conditions and diseases."

The present communication deals with Raynaud's phenomenon on a traumatic basis and resulting from a non-united fracture of the navicular bone. It is presented to call attention to a somewhat unusual complication resulting from fracture of a carpal bone. It may further afford an opportunity for considering one type of mechanism precipitating Raynaud's phenomenon under conditions simulating an experimental setup as presented by a simple, well localized form of human pathologic condition.

CASE REPORT

A Norwegian seaman, forty-four years of age, was first seen by the author in August, 1941. His main complaint was pain in the left hand which he stated dated back to 1919. When then in the navy, he fell one day and hit the dorsum of his left hand against the floor. Subsequent x-ray revealed a fracture of the scaphoid bone. A plaster cast had been applied and worn for two or three weeks. The patient was declared unfit for navy service during the next six months. Upon returning to his former duties, he noted pain in the wrist of his left hand. During the first few years this pain was bearable and did not prevent him from performing his work. However, it bothered him, sufficiently to make him seek medical advice on several occasions. Physiotherapy and massage had a temporary effect.

During the last four years the pain in his left wrist had greatly increased in severity. Consequently, he had been able to carry out his work aboard ship with only the greatest difficulty. During the last two years additional symptoms had been observed; when performing certain functions involving the left hand (e.g., lifting a heavy trunk with this hand), he would note the onset of a tingling sensation of the fingers of this hand, quickly followed by severe pain in the wrist and entire hand, and later a sudden feeling of weakness in the muscles of the hand which would result in the dropping of anything he might be carrying in this hand. During these episodes the fingers of the left hand would appear "completely white and dead," and would feel cold to the touch. A pronounced numbness of the volar sides of the fingers would be experienced. After a few minutes the white color of the fingers would change to bright red or bluish red. Simultaneously he would note a sudden sensation of local heat. The superficial sensitivity and the mobility and strength of the hand would then gradually return. It could not be ascertained if these attacks had any relation to the environmental temperature as they occurred just as frequently during summer as during winter. They were not precipitated by worry or anxiety alone. Similar changes in the right hand or in any other part of the body had never been experienced. The patient also had noted a less

severe pain radiating from the left wrist to the lateral side of the elbow. This pain always occurred with the previously described attacks; however, it would also be present independent of the attacks. Almost every day during the last year he had felt a sore ache around the lateral left epicondyle which was very tender to pressure. Occasionally, he also had noted a moderately large, tender, swelling around the left epicondyle. The patient had grown increasingly nervous and mentally exhausted during the last year due to his physical sufferings, and to the anxiety of life aboard a ship that was constantly travelling through U-boat infested waters.

Physical examination revealed that the patient was well developed and of powerful musculature. He appeared tired and nervous with moderate tremor of the fingers and of the tongue. The thyroid gland was not enlarged. The basal metabolic rate was within normal limits. His blood pressure was 130 mm. Hg systolic and 90 diastolic, with a pulse rate of 74 beats per minute.

Local examination revealed normal mobility of the left wrist and fingers except for moderate inhibition in radial flexion of the wrist. No definite motor or sensory changes were found. Palpation of the left wrist revealed a bony, hard irregular prominence in the bottom of the "snuff box." It was very tender to direct pressure and moderately mobile. Pain in this arm could also be elicited by radial deviation of the wrist joint. This pain would radiate along the radial side of the forearm to the lateral epicondyle of the elbow. Palpation of the left radial and ulnar arteries revealed normal pulsations as was also found in the other peripheral arteries. Physical examination otherwise was essentially negative. Routine laboratory investigations, including the Wassermann reaction, revealed nothing of particular significance.

X-rays revealed a transverse, non-united fracture of the left scaphoid bone. (Fig. 1.) The distal fragment showed a bulging of its anterior, external portion which impinged against the external portion of the articular surface of the radius. This had caused a bony lip formation of the styloid process of the radius at the point of contact with the fragment.

In an attempt to reproduce the previously described attacks of pain and other phenomena the following investigations were undertaken: Insertion of the patient's left hand in ice water

Fig. 1. X-rays of the left wrist before operation.

(8°c.) for ten minutes failed to produce any changes different from that of the right hand, and without any indication of Raynaud's phenomenon. Alternate closing and opening of the left hand for five minutes at a time, with digital pressure compressing the ulnar and radial arteries separately as well as simultaneously, failed to produce any other symptoms than a feeling of muscular tiredness in the hand. By carrying and manipulating a weight in his left hand (a 16 pound typewriter) the patient soon experienced a severe pain in his left wrist; this was associated with a tingling in and pronounced pallor of the fingers. The tactile sensitivity on the ulnar aspect of the second to the fifth finger and over the dorsum of the distal two-thirds of the same fingers was definitely impaired. Simultaneously, the strength of his left hand, as tested by grip of the hand, was greatly impaired as compared to its strength before the carrying of the weight. The pallor persisted for almost one minute after release of the weight and was followed by a sensation of increased local warmth and rubor. Palpation of the pulsation of the radial artery during the stage of pallor indicated a definite decrease although not complete cessation of pulsation. The above experiment with the carrying of the weight in the left hand was then repeated after injection of 8 cc. of 1 per cent

FIG. 2. X-rays of left wrist after operation.

solution of novocain around the palpable movable fragment and around the radial artery of the snuff box. During this experiment the patient did not complain of pain. Likewise, the Raynaud's phenomenon could not be elicited.

Operation was performed on August 24, 1941, using local infiltration with 1 per cent novocain. A 3-cm.-long incision was made in the left snuff box and parallel to the ulnar aspects of the long extensor pollicis tendon. The radial artery was readily located; this was carefully exposed; its wall appeared normal with normal pulsation throughout the exposed segment. A careful palpation of this segment did not give any suggestion of thickening of the wall of the artery nor of the presence of intraarterial thrombus formation. With full extension of the wrist and fingers, the movable fragment of the navicular bone could hardly be felt. Upon maximal ulnar flexion of the wrist, this movable fragment was felt to protrude distinctly and was seen to impinge gently against the posterior wall of the artery. This impingement did not result in any visible or palpable changes in the pulsation or the caliber of the artery. Raynaud's phenomenon of the fingers was not seen to ensue. By gently pulling the

artery and the tendons to one side the movable fragment of the navicular bone was readily freed and removed.

An area of numbness at the base of the left thumb was noted immediately after the operation; otherwise, there were no sensory or motor nerve disturbances. The postoperative course was uneventful with primary healing of the wound. Two days after the operation it was noted that the previously observed local tenderness about the lateral epicondyle of the left elbow had disappeared. On the same day the patient stated that the subjective symptoms of pain in the elbow had disappeared. (Fig. 2.)

Because of the patient's continuous service aboard ship during the war, he was granted a convalescence of five weeks before returning to his ship. During this period further observations with or without carrying a weight in his left hand revealed return of full and normal mobility without pain. Significantly, the Raynaud's phenomenon as observed before operation could not be produced.

Since the patient signed aboard ship again, he has been seen by the author on several occasions, the last time in February, 1946. According to him, he never thinks about favoring his left hand any more and considers it as strong and as useful as before the accident in 1919. Since dismissal from the hospital, no further Raynaud's phenomenon has appeared. Repeated investigations of his peripheral vascular tree have not revealed the presence of any peripheral vascular disease.

COMMENTS

The primary lesion in the present case appears clear enough, namely, a non-united fracture of the left navicular bone of more than twenty years' duration.

The question may be raised: did the patient, aside from the fracture, have a primary peripheral vascular disease? Without taking up a detailed discussion of this point, I believe the report as presented includes essential information sufficient for the conclusion that any type of primary peripheral vascular disease did not exist. The local peripheral vascular phenomenon is therefore concluded to have taken place on the basis of the existing non-united fracture.

How was the Raynaud's phenomenon produced in this case? This seems a simple enough question. To students of the physiopathologic conditions of peripheral vascular diseases Raynaud's phenomenon, however, represents one of great controversy. This is well illustrated by the present conception of the physiopathologic disorder of Raynaud's disease. According to one school of thought, the essential abnormality is to be found in the vasomotor system, this system being abnormally stimulated by extrinsic factors (cold) resulting in hyperactivity of the vasoconstrictor fibers. Opposing this view is the conception that the vasoconstriction primarily is the result of changes in the vessel wall itself.

Without taking up this controversy of the mechanism of Raynaud's phenomenon on a general basis, it may be permissible briefly to discuss it in relation to the present observations. Cold is generally accepted as one of the most common factors eliciting or aggravating Raynaud's phenomenon; this fact naturally does not indicate whether or not the abnormal changes are present primarily in the vasomotor fibers or in the arterioles themselves. It only indicates that the nerve fibers or the arterioles are in a pre-activated state and respond with constriction upon stimulation by low temperature. In the case herein reported the cold factor failed to stimulate the vessels to spastic contractions; in other words, the vessels acted as if no preliminary state of hyperirritability of nerve fibers or arterioles existed.

"Working" of the hand with digital compression of the radial artery failed to produce Raynaud's phenomenon. This would indicate that the phenomenon was not produced by intermittent occlusion of the radial artery itself. Further support for this view was gained by the observations at the time of operation that even with maximal ulnar deviation of the wrist the loose fragments of the navicular bone in no way did result in segmental compression of the radial artery. The logical inference from the latter observation is that we are confronted with a true intermittent constriction of the arterioles distal to the level of the fracture fragment.

Severe pain as well as the typical Raynaud's phenomenon was reproduced in the patient when he carried a relatively heavy weight in such a manner that alternate ulnar and radial flexion would take place in the wrist. From observations made at the time of operation it is possible in retrospect to reconstruct what was taking place during the act of weight carrying. The loose navicular fragment would alternately be expressed and depressed. During its stage of protrusion it would be forced up against the posterior wall of the radial artery. Soon severe local pain and Raynaud's phenomenon would ensue. In the absence of palpable thrombus formation or demonstrable local arterial changes an intimal lesion may still have been present. Only a segmental resection of the artery at this point would definitely have settled the latter possibility. Of significance may be the observation that pain as well as Raynaud's phenomenon during weight carrying could be prevented by preliminary local infiltration with novocain. A possible explanation is that the anesthetic agent interrupted the physiologic action of the sympathetic fibers of the arterial segment thus preventing distal vasoconstriction. On the other hand, without local infiltration the protruding fragment of bone would mechanically stimulate the sympathetic fibers of the arterial segment resulting in vasospasm.

Would the abolition of pain in itself in the aforementioned observations be sufficient to explain the prevention of vasospasm? In other words, can the pain factor in itself lead to Raynaud's phenomenon? In the present case the pain may have been originated by local irritation of the nerve endings in the adventitia of the artery by impingement from the side of the protruding osseous fragment. It may more probably originate from periosteal areas during the expression and depression

of the loose navicular fragment. Irrespective of how the pain originated, the fact remains that pain was a pronounced symptom during weight carrying and, as was expected, disappeared upon local infiltration with novocain.

By closer consideration there appears to exist no need physiologically for a differentiation of vasospasm as caused by mechanical stimulation of adventitial constrictor sympathetic fibers as compared to that resulting from a reflex mechanism on the basis of pain. According to Ranson and Billingsley,[2] "vasoconstrictor fibers are not specific but are identical with those which subserve protopathic sensibility (pain, extremes of temperature)."

If this is so, one may ask why in our case immersion of the hand in cold water did not produce Raynaud's phenomenon? One may postulate differences in the intensity of the stimulus as represented on the one side by the cold factor and on the other by pain produced by weight carrying. It is to be recalled also that the pain associated with weight carrying appeared in a situation with increased local metabolism due to muscle action.

The discussion has been developed to this point as it affords an opportunity to draw parallels from the present simple case to more complex diseases associated with vascular phenomena. The problem of the cervical rib and scalenus anticus syndrome may be considered as an example.

In an occasional case the vascular phenomenon associated with cervical rib or scalenus anticus syndrome may be predominating. Intermittent Raynaud's phenomenon may be present, together with absent or diminished pulsation of the peripheral arteries of the affected arm, leading to trophic disturbances and occasionally to gangrene of the fingers.

An extensive discussion has centered on the physiopathologic disorder of these vascular manifestations. For the sake of simplification two extreme conceptions may be mentioned here. The one represents what may be termed a purely mechanical view, according to which the vascular symptoms derive from simple compression of the subclavian artery by the cervical rib and/or the contractions of the scalenus anticus muscle. According to the other point of view, the vascular changes result from an arterial vasoconstriction due to stimulation of sympathetic fibers of the perivascular or the brachial plexuses. As will be noted this controversy is identical to that around which the discussion has been centered in our case. This is only natural since, by closer considerations, the existing pathologic change in many respects is similar. This similarity consists in an intermittent abnormal relation between a main artery or nerve plexus and the skeletal muscle system.

Granted that the anatomic-pathologic similarity may be striking, still it is not permissible to conclude that the physiopathologic condition would also be identical. However, it may be of some consequence to keep in mind a certain pattern of vascular reactibility taking place under reasonably well controlled and simplified conditions as demonstrated by our case. This pattern is characterized by arterial and arteriolar constriction resulting from mechanical vascular irritation and most probably due to stimulation of the perivascular sympathetic plexus. In the investigative approach to vascular abnormalities in cases of cervical rib such a pattern of vascular reactibility may be worth while keeping in mind.

SUMMARY

A report has been presented of a patient who exhibited a non-united fracture of the navicular bone of twenty-two years' duration, associated with intermittent Raynaud's phenomenon of the corresponding hand. Functional restoration of the hand followed upon operative removal of the projecting movable fragment.

Preoperatively, the Raynaud's phenomenon could be reproduced by weight carrying but not by exposure to cold. Its occurrence

could be prevented by local infiltration of novocain around the loose fragment.

The discussion essentially centers on the mechanism of Raynaud's phenomenon as produced by this relatively simple lesion and under reasonably well controlled clinical conditions. It is concluded that in all probability Raynaud's phenomenon in the present case resulted from stimulation of the perivascular sympathetic plexus leading to vasoconstriction of the distally located arteriolar branches. It is implied that this represents one simple mechanism

explaining Raynaud's phenomenon. Others definitely exist. Certain parallels are drawn to the more complex problem of vascular phenomena noted in cases of the cervical rib and scalenus anticus syndrome.

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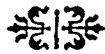
A. H. BAKER reports the case of a four year old patient in whom several diagnostic lumbar punctures were performed. The child developed backache and then difficulty in walking and sitting. The two intervertebral spaces between the second to the fourth lumbar vertebrae later became narrower, according to x-rays, and it is believed that probably the lumbar puncture needle ruptured the annulus and permitted the liquid nucleus pulposus to seep out through this perforation. In adults lumbar puncture is less apt to be followed by such an unusual complication because the nucleus pulposus is more solid in such cases. It is well to keep this possible complication in mind when one is required to perform lumbar punctures in infants and children. (*Richard A. Leonardo*, M. D.)

CYSTIC HEMANGIOMA OF THE SPLEEN

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CAPILLARY and cavernous hemangiomas of the spleen constitute a relatively rare pathologic entity. Most reported cases[1-3] have been from autopsy findings in which an incidence of about 0.1 per cent has been recorded. A few cases have been discovered at operation which has usually been performed for hemorrhage following trauma.[4,5] Hemangioma and lymphangioma are of particular interest in the present report because they may be the underlying basis for cystic changes giving rise to a variety of pathologic changes in the spleen.

In general, it may be stated that cysts of the spleen constitute a pathologic curiosity and are extremely rare in clinical surgery. McClure and Altemeier[6] report 148 splenic cysts in a review of the literature up to 1941. As a cause of splenomegaly, cystic disease is responsible for a very low percentage and, apart from parasitic invasion (e.g., echinococcus disease), only 108 splenic cysts were reported up to 1941 by Watts and Warthen.[7] Deneen[8] states that a review of the Bellevue Hospital statistics from 1904 to 1942 revealed no case of hemorrhagic cysts of the spleen with a similar record at St. Vincent's Hospital for the period 1910 to 1940. McClure and Altemeier[6] add to their report a recent case of a patient operated upon by them at the Henry Ford Hospital. Paul[9] believes that cysts of the spleen are very rare. He states that Andral reported the first case in 1829 and he then reports an operative case in which the spleen presented one large and several smaller cystic areas. In his case there was a history of malaria and in the pathogenesis of this lesion an antecedent malarial infection with trauma appear to be prominent features. Andral's case microscopically presented

cyst walls lined by stratified epithelium. The same writer presents a simple classification of splenic cysts as follows: (1) Hydatid cysts of the spleen; (2) multiple serous cysts associated with polycystic disease of the kidneys; (3) single cysts consisting of (a) dermoid cysts, (b) epidermoid cysts and (c) serous and blood cysts devoid of an epithelial lining and (4) Multiple serous cysts.

McClure and Altemeier[6] present a more elaborate classification: (1) true cysts—lined by a specific secretory membrane; (a) epithelial consisting of dermal and epidermal; (b) endothelial consisting of lymphangioma, hemangioma, polycystic disease and some serous cysts; (c) parasitic—lined by protoplasmic matrix containing numerous nuclei making up x-hydatid cysts. (2) false cysts—no specific secretory lining making up (a) hemorrhagic; (b) serous; (c) inflammatory with acute necrosis in infection and chronic tuberculosis; (d) degenerative liquefaction of infarcted areas caused by embolism or arterial thrombus.

The subject of the present report appears to have true cysts of endothelial origin. Gradations and changes in the pathologic picture, depending upon the age of the lesion, areas studied, concomitant disease, etc., makes overlapping of classification likely. Sweet[10] like most writers emphasizes the relative paucity of clinical symptoms associated with splenic cysts. Apart from the acute picture of shock and hemorrhage which may occur with rupture, the chief complaints are those referable to the presence of a mass in the hypochondrium; because of its size, this may give rise to varying degrees of discomfort and dysfunction due to pressure on neighboring viscera. Differential diagnosis of splenomegaly ordinarily presents no great diffi-

culty but such conditions as polycystic kidney, retroperitoneal blastoma, mesenteric cysts, etc. must be considered either alone or in association with splenic cysts. The diagnosis of cystic disease *per se* must usually await surgical exploration or necropsy although Sweet and others have pointed out the value of x-ray studies.

Because of the benign nature of the process, splenectomy for splenic cysts gives excellent results. However, the technical problems involved in splenectomy for any splenomegaly present a wide range, and in some cases they may be of such a nature as to preclude successful surgical extirpation of the tumor. These problems have been reviewed by Balfour,[11] Henry[12] Maingot,[13] Behrend[14] and many other surgeons. Recently, Dunphy[15] summarized surgical problems, reviewed surgical anatomy and presented some very important points of great technical significance in splenectomy. We agree with him that splenectomy is often a relatively simple procedure in the case of a small spleen; here, even in the presence of severe trauma and hemorrhage, proper preparation coupled with good anesthesia will ensure a fairly simple and successful operative procedure. However, in long-standing pathologic states (Banti's syndrome, splenic anemias, cirrhosis, etc.) the development of numerous extremely vascular adhesions, the great friability of the spleen and the technical obstacles presented by the size of the organ and its fixation to adjoining viscera (especially the stomach, colon and diaphragm) may combine to render splenectomy extremely hazardous or impossible even for the most experienced and skilled surgeon.

The present case is deemed worthy of presentation not only because of the rarity of the condition but also because of the associated pregnancy, the long history, the striking surgical and pathologic findings and also to reemphasize the surgical difficulties which one may anticipate. We believe it also serves to point out the great value of adequate pre- and postoperative care.

CASE HISTORY

Mrs. B. C. (Hospital No. 7752), a married Negro female, twenty-five years of age, was first admitted to the out-patient service, Herrick Memorial Hospital, April 29, 1946. Her chief complaint was a painful mass in the left hypochondrium of about seven month's duration. Her family history revealed the following: the medical status of her parents was unobtainable; there were several siblings; there was no known history of tuberculosis, cancer or other familial diseases. In regard to the patient's past history she denied that there were any serious illnesses or operations. The patient, on closer questioning, stated that she may have had malaria during her childhood in the South, and also stated that she had "spleen trouble" at the age of thirteen. Catamenia began at the age of 15; her periods were regular every twenty-six days, lasting for three days; there was a moderate flow and no dysmenorrhea. The patient had been married five years. She has two living children; she had one spontaneous miscarriage in 1944. Last menstrual period was on March 13, 1946. She had been troubled by nausea and painful breasts since the onset of her present amenorrhea; she thought she was probably pregnant.

Physical examination revealed a fairly well nourished, young colored woman. Her weight was 143 pounds and blood pressure, 90/60. System findings were unimportant except for abdominopelvic findings. There was a large, irregularly round, firm, slightly tender mass occupying the left hypochondrium. It extended from below the rib cage to the left flank, well below the umbilicus and mesially to the right of the midline. A notched surface was palpable. The liver was not enlarged to percussion in the right hypochondrium and dullness merged with that of the mass on the left side. Abdominopelvic examination showed the uterus to be uniformly enlarged, soft and extending above the symphysis; the mucous membranes of the vault were dusky.

Clinical diagnosis: Early pregnancy; splenic tumor.

X-ray and laboratory findings revealed that a plain film showed extreme enlargement of the spleen; the left costophrenic angle was compressed and there were old, healed tuberculous foci in the left lung; the finding suggested leukemia or other lymphoblastoma. Urinalysis

was within normal limits; repeat blood counts showed mild secondary anemia and moderate leukopenia with normal distribution of leukocytes; fragility tests were within the normal range; platelets, 249,000; blood was group III-B and Rh positive.

Tentative diagnosis: Splenomegaly of unknown etiology, complicated by pregnancy.

The patient was admitted to the hospital July 22, 1946. In view of her generally good condition it was decided to postpone transfusion until surgery and thereafter as a difficult operative course was anticipated. She was operated upon on July 23, 1946, under nitrous-oxide-oxygen ether. Saline was commenced before the abdomen was opened and this was followed with two units of whole blood. A left rectus incision extending from the costal margin to the superior border of the ilium was made. As soon as the abdomen was opened 25 mg. of intocostrin were administered by injection into the perfusion fluid. On opening the peritoneum the spleen presented as a huge cystic mass widely displacing the stomach and the colon. A large knob-like tumor or cyst occupied the lower pole of the spleen and the upper surface was deep under the dome of the diaphragm. There were whitish somewhat raised areas scattered over the remainder of the surface, and the organ was densely adherent to the diaphragm, liver, lateral abdominal wall, transverse colon, stomach and the omentum. These adhesions were very thick, tough and extremely vascular. The splenic tumor was completely immobile until dissection of its attachments was commenced. The gastro-splenic omentum was doubly ligated as high up as possible in serial segments with 2 chromic and carefully cut between ligatures. Because of very dense adhesions in the lesser sac, exposure and control of the major vessels through this approach did not appear feasible. Freeing of adhesions was therefore proceeded with, using sharp and blunt dissection of small zones with secure hemostasis at each step. The peritoneum of the left parietal region above the lienorenal ligament was carefully incised and stripped down with gauze. The right hand was then insinuated between the upper pole of the mass and the diaphragm; here blunt dissection of adhesions had to be accomplished blindly. It was then found possible to push the huge splenic tumor into the wound after which mesial rotation exposed the broad pedicle with

the main blood vessels. These were carefully isolated and doubly ligated with No. 4 chromic, a double clamp was placed distally and the pedicle cut through. The pedicle was then transfixed and tied with a third ligature which was then tacked down to the splenic bed. As soon as the spleen was delivered two large hot saline packs were snugly applied to the tumor bed. It was remarkable and very gratifying to note, upon removal of the packs, that the field was dry. The opening in the gastrocolic omentum was repaired with plain o catgut. The abdomen was closed in layers with interrupted sutures to muscle, fascia and skin and with four heavy silk retenion sutures. The patient's condition was excellent during the entire procedure. Systolic blood pressure ranged from 120 to 110 and diastolic pressure from 80 to 60 mm. The pulse averaged 90 and respirations 20. The entire procedure took one hour and forty-five minutes. On returning to the ward the patient was given another unit of whole blood, and later that day 1,000 cc. of 10 per cent glucose in saline. The postoperative course was remarkably uneventful. The highest postoperative temperature was 101°F. for one day and the pulse and respiration remained within normal limits. She was taking liquids on the first postoperative day, a light diet on the second day and was ambulatory and on a general diet on the fourth postoperative day. The patient complained of some back pains and pelvic discomfort resembling labor pains on the ninth day; these subsided with sedation. She left the hospital on the twelfth day. The stitches were removed and the wound appeared to be healing nicely per primam. She reported subsequently to the obstetrical service of the out-patient department and pursued an uneventful prenatal course. On November 27, 1946, she was delivered with low forceps of a 7 pound baby girl. Her puerperal status and course were entirely uneventful. The patient has reported subsequently to our Clinic. She was reviewed December 30, 1946, at which time she was feeling fine and had no further complaints.

Pathological report revealed the following: specimen was labelled 'spleen.' It was a large, dusky, cystic mass, measuring about 22 by 15 by 8 cm. On cut sections it appeared very congested and was riddled by variously sized cystic cavities, these varying in size from a few mm. to several cm. in diameter. Microscopic examination of sections from different areas
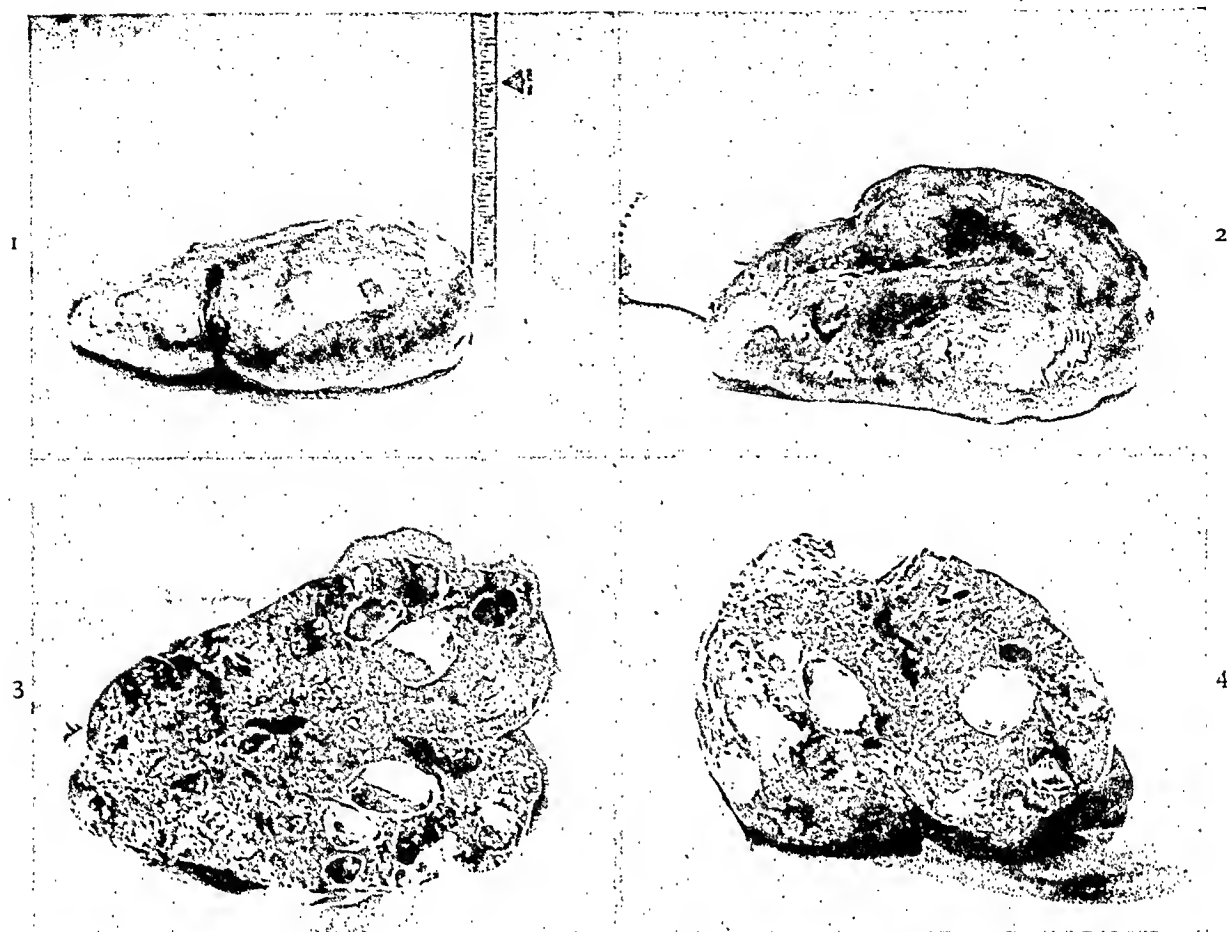
FIG. 1. Superior and lateral surface of splenic tumor; relative size indicated.
FIG. 2. Inferomesial surface showing cystic masses.
FIG. 3. Longitudinal section showing large and small cysts.
FIG. 4. Cross section showing cysts and heavy scarring.

were examined. There was almost complete destruction of the splenic pulp. This had been replaced by broad bands of fibrinous and fibrous tissue which appeared to surround vascular channels lined by endothelium. In some areas a capillary hemangiomatous network was well demonstrated. There was no evidence of malignancy. In other levels the general pattern of a plexiform hemangioma was similarly noted. Wide trabeculae dominated the picture with small islands of blood vessels spreading and compressing the tissue. In the larger spaces a liquefaction necrosis had converted the picture from red blood cells and vessels into a homogeneous acidophilic matrix.

Diagnosis: Cystic degeneration in plexiform hemangioma of the spleen. (Figs. 1, 2, 3 and 4.)

SUMMARY

The clinical history which went back to early childhood is extremely interesting. Also notable is the relative paucity of complaints referable to the growth until

it had reached huge proportions. The possibility of a long-standing latent malarial infection may be inferred although no hematologic or pathologic evidence was found in our study. It is interesting to speculate on the possible rôle of trauma in the initiation of hemorrhages and cystic degeneration in an organ which is the site of capillary and cavernous hemangiomas. These conditions would undoubtedly render the spleen unusually susceptible to injury. Again, however, no definite history of trauma was elicited but forgotten injuries may well have occurred repeatedly during the life of the patient. A comparison of the histologic features of our case with those we have had the opportunity to review in the literature leads us to remark on the unusually striking features of hemangioma depicted in this case. The association of a relatively advanced pregnancy as a troublesome complication is of

clinical interest, and the favorable outcome for mother and child indicates to what a degree nature will tolerate a pathologic state and a surgical insult of great magnitude without relinquishing its purpose.

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CANCER of the Islets of Langerhans is a fast-growing tumor, occurring usually in the tail of the pancreas of middle-aged men, with or without hypoglycemia. Unless metastases are discovered, usually in the liver and neighboring lymph nodes, the malignant nature of the growth is likely to escape detection by the surgeon. (*Richard A. Leonardo*, M. D.)

EWING SARCOMA OF THE RIB*

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TUMORS of the chest wall are not rare and, in this group, neoplasms of the costal cage are among the most frequent in occurrence. However, it has been quite unusual to encounter a Ewing sarcoma of a rib, and for that reason this report is being made. The tremendous size of the tumor in the case to be reported constituted an additional cause for this contribution.

A brief and incomplete review of the literature for the past ten years reveals reports of 311 cases of Ewing's sarcoma in five groups of appreciable size.[1-5] Twenty-one of these were designated as primary tumors of the ribs, a total of 6.7 per cent of the five groups of cases. Only one patient was reported to be alive when the various articles were prepared for publication. It is generally accepted that rib tumors of this type are extraordinarily serious with a rapid course and an unfavorable end result.

In general, more than half the cases of Ewing's sarcoma occur before the twentieth year, about 80 per cent before the thirtieth year, and it is rare after the fortieth year of age. More than 70 per cent of the cases are in male patients. Additional tumors of bone soon make their appearance, especially in the base of the skull, pelvis, ribs and vertebrae. Whether these are metastatic tumors or secondary tumors of non-metastatic origin is a point which has been subjected to debate. These usually appear towards the end of the first year or earlier and are often seen first in the skull. In many cases the regional lymph nodes become involved. Pulmonary metastases occur in nearly all cases but they appear late in the course of the disease in contradistinc-

Fig. 1. Chest film revealing the great size of the intrathoracic position of tumor.

tion to osteogenic sarcoma which metastasizes to the lungs earlier and in a more prominent fashion. In late stages nearly the entire marrow system is the seat of tumor tissue with extensive destruction of bone.

Diagnosis is aided by microscopic examination of tumor tissue, the radiographic characteristics of the lesion and by the typically prompt effect of roentgenotherapy upon the tumor. There is, of course, considerable contention as to whether Ewing's sarcoma is an entity, and among those who believe that it does exist, there is disagreement over the cellular origin of the tumor.

Treatment of the original lesion has been accomplished by the use of x-ray therapy, Coley's toxin, curettage, resection or a combination of these methods. The results have been far from satisfactory.
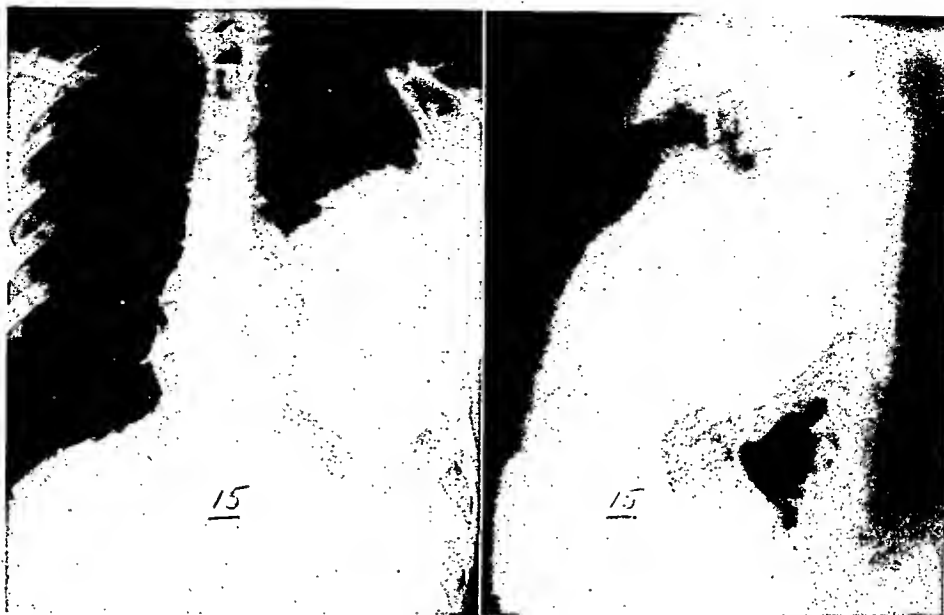
FIG. 2. Chest film taken five days after institution of x-ray therapy. The effect was prompt and is already quite dramatic.

FIG. 3. Lateral view of chest taken at time Figure 2 was made.

CASE REPORT

A nineteen year old Marine was admitted to the hospital on December 16, 1944, complaining of deformity of the left chest wall and of left-sided pain. The pain had begun one year before and had persisted since onset. The deformity had been noticed by the patient five months before admission and had become progressively more marked.

Physical examination immediately revealed a marked bulging deformity of the left chest wall, chiefly over the anterolateral aspect. The mass was hard and non-tender, poorly defined at the borders and of a large size. Chest x-ray films revealed an enormous intrathoracic extension of the mass. (Fig. 1.)

An aspiration biopsy was performed and a diagnosis of Ewing sarcoma was made. Intensive x-ray therapy was instituted and a total of 5,000 r given, employing 400,000 KV through a single large portal which encompassed the entire extent of the tumor. The radiotherapeutic response was almost immediate after the initiation of treatment as can be seen in Figures 2 and 3. These x-ray films were made only five days after the institution of radiotherapy and a marked reduction in the size of the tumor had occurred. Within ten days, the visible chest wall deformity had disappeared, and at the end of three weeks, the tumor had become much smaller as can be seen in Fig. 4. At this time, considerable destruction of the mid-portion of the left fifth rib could be seen on the x-ray films, indicating that this bone had been the primary site of the neoplasm. The roentgenotherapy had been completed very shortly afterward but no further reduction in size of the tumor could be demonstrated in the ensuing three weeks. Accordingly, the decision was made to resect the remaining tumor mass.

On February 21, 1945, the operation was performed. The major portions of the fourth, fifth and sixth left ribs were removed along with the intercostal structures of the third, fourth, fifth and sixth intercostal spaces. Gross evidence of invasion of the left lung was present. Consequently, about one-third of each of the left pulmonary lobes was resected and the entire specimen was removed *en masse*. The defect in the thoracic cage was necessarily rather large and reconstruction of the boney chest wall by means of tailored ribs of tantalum was considered but the plan was rejected because of the possibility of poor wound healing as a result of the effect of the x-ray treatment upon the soft tissues of the thoracic wall. There had been considerable effect upon the skin. As a result, a minimum of buried sutures were employed in the chest wall closure and retention sutures were inserted to suplement the closure.
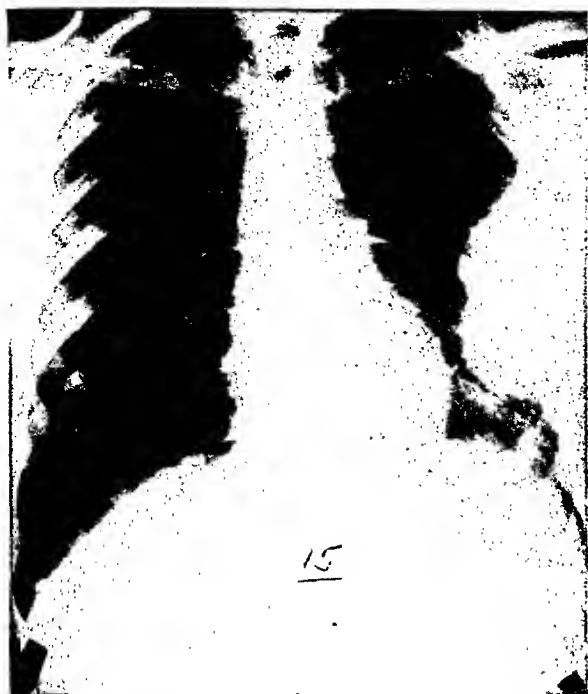
FIG. 4. Chest film taken near the conclusion of x-ray therapy. The reduction in size had taken place over three weeks time but no further effect was noted during the succeeding weeks.
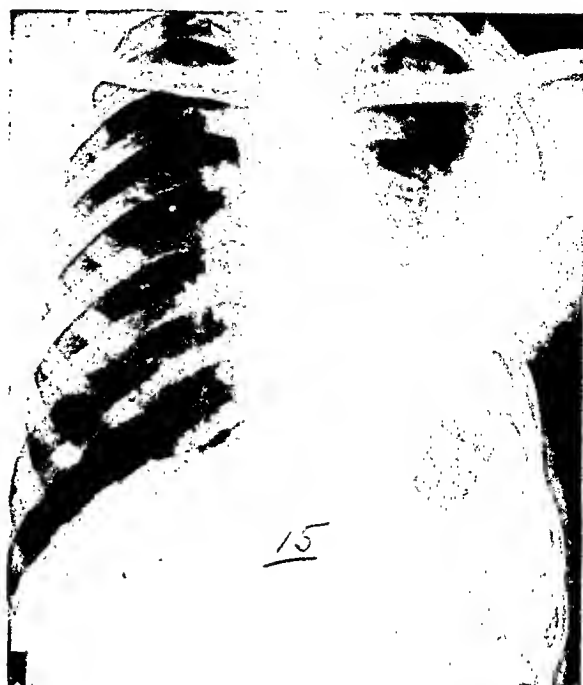
FIG. 5. Chest film made three months after operation. The residual defect in the costal cage is visible. The diaphragm has become elevated. The patient was not short of breath upon exercise.

A catheter was left within the pleural space and brought out through the anterior portion of the wound. This was connected to a water-seal bottle and it was employed to facilitate expansion of the partially resected pulmonary lobes and to permit drainage of blood and fluid from within the thoracic cavity. The catheter was removed on the fourth day. The wound healed promptly except for a small portion of the skin at the anterior limit of the incision which required three and one-half weeks for complete healing. The patient began to get out of bed on the seventh postoperative day but his return to normal was prolonged over a period of one month. During this period there were no evidences of cause for alarm other than a continued slow loss of weight, failure to regain appetite and a discouraging delay in regaining strength and his preoperative state of morale and spirit. However, the improvement in all phases was very rapid after it began and a very satisfactory recovery took place.

A considerable defect in the boney thoracic cage resulted as can be seen in Figure 5, a chest film made three months after operation. There was surprisingly little visible deformity of the chest wall as a result, but the patient was somewhat apprehensive over the loss of the protective function of the missing ribs, particularly anteriorly over the cardiac area. Accordingly, a specially fitted protective device was

made which was both comfortable to wear and easily retained in place by two straps. The patient was very well satisfied with the appliance and wore it regularly.

Examination of the surgical specimen revealed that the tumor had arisen in the fifth rib which was partially destroyed, and that it had invaded the attached portions of both pulmonary lobes. Microscopic and gross examination indicated that the invasion had not extended to the line of pulmonary resection. The microscopic picture revealed that the tumor cells had suffered too many destructive changes from the x-ray therapy to permit identification or to allow comparison with the tissue obtained by aspiration biopsy.

Thirteen months after operation the patient was well and without evidence of distant or local recurrence.

SUMMARY

1. A case of Ewing sarcoma of the fifth left rib is described.

2. The tumor had reached astonishingly great size before the patient was received for treatment.

3. The diagnosis was made by aspiration biopsy and supporting evidence is afforded by the rapid and very marked radiosensitivity displayed by the tumor.

4. When a maximum of effect seemed to have been achieved from radiotherapy, a radical resection of the tumor with adjacent chest wall structures and pulmonary tissue was performed.

5. The patient was well thirteen months after operation but the prognosis is very doubtful at best.

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ACCORDING to S. Annersten, scalenus anticus syndrome is often due to pressure upon the brachial plexus by this muscle, especially when the costotransverse process of the seventh cervical vertebra is unduly large and that often no distinct cervical rib is present. In such cases simple scalenotomy is all that is needed to relieve the patient of his or her symptoms. (*Richard A. Leonardo*, M. D.)

PRIMARY TORSION OF THE OMENTUM*

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PRIMARY torsion of the great omentum is one of the infrequent acute surgical conditions of the abdomen. The literature records but seventy cases. The condition has seldom if ever been correctly diagnosed prior to operation. In nearly all the reported cases the preoperative diagnosis has been acute appendicitis since the clinical symptoms of the two conditions are similar.

Torsion of the omentum means a twisting or rotation of part or all of that structure upon its axis. An ovarian cyst becomes twisted upon its pedicle in the same way.

For many years no attempt was made to distinguish between this type of torsion and that which is commonly observed to occur in hernia or as a result of neoplasms, adhesive bands or inflammatory processes. It remained for Eitel in 1899 to report the first case of primary intra-abdominal torsion occurring independently of these etiologic factors. Later cases were reported by Baldwin and by Syme in 1902 and by Scudder in 1904.

In 1938 Teller and Baskin clarified the subject by presenting the following classification: (1) primary (pure, true, idiopathic) which constitute simple torsion and torsion with herniation of the mass through the omentum itself and (2) secondary which make up hernial; hernio-abdominal; abdominal; pelvic and combined.

In 1946 Altemeier and Holzer reviewed the literature and found sixty-four cases; their six cases from the Cincinnati General Hospital brought the total to seventy recorded cases. Two more cases are added by this report.

CASE REPORTS

CASE I. G. K., a fifty-two year old, white male, was admitted to the Methodist Hospital January 10, 1939, with a history of gas and bloating for three days accompanied by increasing right abdominal pain. The pain had developed gradually but had remained constant and was without any known cause. It was described as dull and never severe or colic-like. No nausea or vomiting had occurred but there was occasional bitter regurgitation into his mouth.

Examination showed a moderately well nourished middle-aged man obviously in discomfort. His temperature was 100.6°F; his pulse was 100. General physical examination was essentially normal except for the abdominal condition. There was slight rigidity and tenderness in the right upper quadrant; this condition was marked in the right lower quadrant, the maximum point of discomfort being at McBurney's point.

Laboratory examination showed the urinalysis to be essentially negative and the white blood cell count to be 13,800.

Diagnosis of acute appendicitis was made and operation was performed under spinal anesthesia. The abdomen was opened through a pararectus incision, and it was at once noted that the appendix was nearly obliterated and was not the cause of the acute inflammatory symptoms. At the same time free sanguineous fluid was observed and about 8 inches of the great omentum just above the appendix was seen to be strangulated and showed evidence of the start of gangrene. This was the result of primary torsion. Because it was impossible to unravel the omentum, the diseased portion was resected at the pedicle. Appendectomy was performed. A search of the abdomen revealed no evidence of other pathologic disease. The patient made a normal recovery and was discharged on the tenth postoperative day.

Pathologic examination showed an omental mass approximately 8 by 6 inches, the blood vessels of which were thrombosed; there was hemorrhagic extravasation, necrosis and gangrene.

For the past fifteen years I have pre-ferred the pararectus to the McBurney incision because it permits better expo-sure of the abdomen for just such un-usual conditions as this or for a difficult appendectomy.

CASE II.   J. S., a white male forty-one years of age, was admitted to the Methodist Hospital December 3, 1942, with a diagnosis of acute appendicitis. For thirty-six hours he had com-plained of a steady, dull pain in the lower right side. There was no diarrhea or constipation but he was nauseated and had vomited once. He complained of marked tenderness on pres-sure. There were no urinary complaints and no history of a chill.

The temperature was 101°F., the white blood count was 17,240 and the urinalysis was negative.

Physical examination was negative except for moderate rigidity and tenderness in the right lower quadrant. The patient's discomfort seemed out of proportion to the clinical find-ings, but the high blood count and poly-morphonuclear count of 72 suggested a prob-able gangrenous appendix.

Immediate operation was advised. Under spinal anesthesia a pararectus incision was made and the appendix located. It was at once evident that this was not the source of the trouble; further search revealed a mass in the right upper quadrant. The incision was extended upward, and the mass was revealed to be a primary torsion of the omentum. The latter was necrotic at the lower 3 inches and showed evidence of marked inflammatory reac-tion and hemorrhage. The diseased tissue was resected at the pedicle, the appendix was re-moved and, further examination revealing no other abnormality, the abdomen was closed. Convalescence was normal and the patient was discharged seven days later.

Pathologic examination showed a necrotic piece of omental tissue approximately 6 by 4 inches with a marked torsion at the neck. The vessels were thrombotic and the tissue showed evidence of the start of gangrene.

ETIOLOGY

The etiology of primary torsion of the omentum is unknown. Nearly all the re-ported cases occurred on the right side, and since this side of the omentum is larger and more mobile than the left side it would be more apt to be involved by exaggeration of the normal movement of the omentum. Some observers, such as Teller and Baskin, thought that obesity might be a factor but neither of the author's cases occurred in obese persons. Others have reported trauma as a possible cause. Payr believed that hemodynamic forces might be important and Sellheim believed that rotary move-ment of the body was a factor. These are all hypotheses and the real cause remains a mystery.

DIAGNOSIS

About the closest that one may expect to come in diagnosing this condition is to state that the patient has an acute surgical condition of the abdomen and should have an immediate exploration. The writer is fairly confident that no one could have correctly diagnosed the two patients he operated upon although one might have hazarded a guess. When one stops to con-sider that about 300,000 appendectomies are performed yearly in this country and that only seventy-two cases of primary omental torsion have been reported, one sees what a long chance one would be taking in naming this condition as a pre-operative diagnosis. The symptoms are nearly identical as are the physical find-ings. It is true that in the second patient I undoubtedly was able to palpate the rolled-up and twisted omentum. I assumed it was just that but I had expected it to surround a gangrenous appendix.

Altemeier and Holzer believe that the progression of symptoms is usually less rapid than in appendicitis. This may be true but it has been the author's experi-ence that in appendicitis the progression of symptoms may be rapid or slow, depending upon the resistance of the individual, the virulence of the organisms and the manner of treatment the patient receives. As these authors so well state, however, it should be emphasized that no practical importance is attached to a preoperative diagnosis of

omental torsion since the similarity of the signs and symptoms to those of acute appendicitis make surgical exploration of the abdomen mandatory.

There are a few important factors about this condition that the writer believes should be emphasized. Because of the rarity of the condition, there is the possibility that it may be overlooked and only a non-responsible appendix removed. The condition is one calling for immediate surgery to prevent the process from becoming extensive and so endangering the patient's life. A thorough resection of the diseased omentum should be performed with careful ligation of the blood vessels. When surprised on opening the abdomen not to find the appendix the cause, bear in mind omental torsion, regional enteritis, Meckel's diverticulum not to mention a host of other conditions. In the acute surgical condition of the abdomen there are surprises that always keep the experienced surgeon in a humble mood.

CONCLUSIONS

Two cases of primary omental torsion are added to the seventy previously reported cases in the literature.

1. The etiology of this condition remains unknown.

2. Primary is to be differentiated from secondary omental torsion. The latter condition is observed to occur commonly in hernias.

3. There are no distinguishing clinical symptoms in this condition.

4. Primary omental torsion closely simulates acute appendicitis and calls for immediate exploratory laparotomy.

5. Only one death resulting from operation is recorded in the literature.

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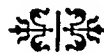
SULFATHALIDINE IN LOW POSTOPERATIVE FISTULAS OF THE ILEUM*

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USE of sulfasuxidine and sulfathalidine[†] in the treatment of surgery of the large bowel has been definitely established. Sulfathalidine or phthalylsulfathiazole, the most recently introduced agent in colonic surgery, was introduced by Poth and Ross[1] in 1943. Poth and Knotts[2] noted that diarrhea and purgatives adversely affected the use of sulfasuxidine in surgery of the colon. The purpose of this paper is to show that sulfathalidine is helpful and of value in postoperative fistulas involving the lower ileum.

Poth[3] and Angelo[4] in their articles summarize the advantages of sulfathalidine as follows: (1) It has a more rapid action on Bacillus coli and the vegetative forms of clostridia; (2) the feces become soft and not fluid; (3) it is effective in diarrhea; (4) no toxicity has been reported as yet; (5) it is twice as potent as sulfasuxidine and so the dose need only be one-half as much.

Poth, Ross and Fernandez[5] working on dogs showed that when sulfathalidine was used preoperatively and postoperatively, the inflammation, resulting from spillage of intestinal contents through the suture line in open anastomosis, subsides and the tissues undergo orderly repair and healing. Whereas 43 per cent of the controls died from generalized peritonitis, none died when the drug was used.

Poth states that in the calculation of the dosage it should not be the weight of the patient, but the inner surface of the bowel area and the rate at which this drug passes over this that determines the quantity necessary to effect a desired action.

The following are two case reports of intestinal fistulas that resulted from intestinal resection. Penicillin and sulfadiazine were used without result. One of the patients had in addition a disruption of the entire wound with multiple fistulas. These patients were put on a dosage of 2 Gm. of sulfathalidine every four hours although Angelo in his series of patients believes that only 5 Gm. in divided doses are necessary.

CASE REPORTS

Case 1. L. B. W., aged thirty-eight, was admitted on August 7, 1946, with a two-day history of nausea and vomiting. This was accompanied with intense colicky pain. A previous operation was done on the gynecological service, March 8, 1946. At that time a right salpino-oophorectomy and left cornual resection was done.

On examination an acutely ill, colored female was seen. All essential findings were localized to the abdomen. The latter was moderately distended with a mass in the lower suprapubic area. The white count was 31,350 with 94 per cent polymorphonuclears. The red blood count was 3,720,000 with 100 per cent hemoglobin and a sedimentation rate of 18 mm. in twenty minutes. The urinalysis was negative.

The patient was taken to the operating room at which time 2 feet of gangrenous bowel (ileum) was removed. An end-to-end anastomosis of ileum over a Murphy button was done because of the extremely toxic condition of the patient; this type of procedure was used because it allowed the surgeon to complete the operation in the shortest possible time.

Postoperatively the patient was put on penicillin, Miller-Abbott tube, Wangensteen drainage, intravenous fluids, plasma and blood. The course was extremely stormy; the patient became delirious and toxic. On August 14, 1946, the patient had a passage of flatus and feces. On August 17, 1946, the patient developed an intestinal fistula. Sutures were removed; the fecal drainage was profuse.

On August 19, 1946, sulfathalidine was started. An initial dose of six Gm. and 3 Gm. four times a day was used. On August 24, 1946,

the fecal drainage was markedly reduced. On August 26, 1946, there was only very slight fecal drainage. On August 29, 1946, there was no fecal material but a slight purulent discharge. It is to be noted that the temperature until the start of sulfathalidine ranged between 101 and 102°F.; since then the temperature has remained flat.

Laboratory findings revealed the following: On August 8, 1946, the white blood count was 31,350 with 94 per cent polymorphonuclears; the red blood count was 3,720,000 with 100 per cent hemoglobin; the sedimentation rate was 18 mm. in twenty minutes; creatinine was 1.3; urea nitrogen was 15.0 and glucose was 76.0. On August 14, 1946, hematocrit was 40.9; sulfa level, trace; total proteins, 5.87; albumin, 2.99; globulin, 2.88. On August 19, 1946, hematocrit was 47.5; albumin, 4.14; globulin, 2.39; total proteins, 6.53; Wassermann, negative; urine, negative. On August 21, 1946, the white blood count was 11,200 with 74 per cent polymorphonuclears. The red blood count was 3,210,000 with 74 per cent hemoglobin. On August 28, 1946 the white blood count was 10,500 with 82 per cent polymorphonuclears and the red blood count was 3,460,000 with 70 per cent hemoglobin.

CASE II. M. G., aged thirty-one, was admitted on June 11, 1946, with a nine-hour history of right lower quadrant pain associated with nausea and vomiting. Her last bowel movement was on the morning of admission via an enema. Neither a previous history of pain nor a previous operation could be ascertained.

On physical examination a young, colored female in good condition was seen. All essential findings were in the abdomen. There a mass was noted extending from the right inguinal region to the umbilicus. This mass was cystic, round, well demarcated and non-tender. On vaginal examination the same cystic mass was in the right fornix. The impression then was an ovarian cyst. Ten hours later the patient complained of pain in the right side. She was cold, clammy and in shock. The impression was a twisted ovarian cyst. On operation about 6 feet of gut was found extending proximally 4 inches from the ileocecal valve. The operation was an ileotransverse colostomy end-to-side, done with a Furniss clamp.

Postoperatively the patient was put on Miller-Abbott tube, Wangensteen suction and was given plasma, blood, intravenous fluids and penicillin. The patient was delirious the first few days. On June 20th, an infection of the lower angle of the wound resulted. The Michel clips were removed and a Penrose drain inserted. On June 28th, the entire wound disrupted down to the peritoneum and a profuse fecal discharge was noted. The patient was continued on penicillin, but the intestinal fistula persisted. The question of side tracking the fistula arose. Meanwhile the patient was put on sulfathalidine, 6 Gm. for the first dose and 2 Gm. every four hours. Seven days later no fecal drainage was noted. The wound granulated nicely.

Laboratory findings revealed the following: on June 13, 1946, creatinine was 1.3; urea nitrogen, 8.0; sugar, 100; Kahn, 4 plus; urine, negative; white blood count, 11,850 with 87 per cent polymorphonuclears and red blood count, 4,300,000 with 78 per cent hemoglobin. On July 29, 1946, creatinine was 1.3; urea nitrogen, 9.0; glucose, 75.0; chlorides, 472; albumin, 4.14; globulin, 2.97; total proteins, 7.11.

In these two cases the clinical condition of the patients was such that it was our opinion and the opinion of several experienced surgeons that further operative intervention was indicated to close the existing fistulas, but after we put the patients on sulfathalidine they closed without further operative procedures.

CONCLUSIONS

1. Sulfathalidine should be tried in all cases of low postoperative fistulas of the ileum before any operative procedure is attempted.

2. Sulfathalidine's antiseptic action is apparently effective in the lower ileum as well as in the colon.

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MESENTERIC THROMBOSIS OF LOWER ILEUM FOLLOWING RESECTION OF THE SIGMOID COLON FOR CARCINOMA

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IN the case to be described resection of the sigmoid for obstructing carcinoma was followed by mesenteric thrombosis in the terminal ileum. Following successful resection of 14 inches of ileum, obstruction due to adhesions developed, necessitating a third laparotomy. Recovery ensued. We think this sequence of events is interesting enough to warrant a brief report:

CASE REPORT

Mr. H. M., aged fifty-eight, was first seen June 6, 1947. He had bleeding per rectum for four or five months which was apparently no worse in the past week than it was before. For the past month he had passed more gas than usual and for this length of time there had been frequent, small bowel movements and some "gurgling" in the abdomen. Also during the past month there had been pain in the left side of the abdomen, intermittent in character and not severe. This was always better following a bowel movement.

Examination revealed a well developed man, rather small of stature. General examination was negative except for some slight tenderness in the left side of the abdomen and some suggestion of gas. X-ray showed a definite filling defect in the mid-sigmoid. Preoperative films also showed a rounded, soft tissue mass in the region of the bladder which the roentgenologist thought might be a distended bladder.

After careful preoperative cleansing, oral sulfasuxidine, etc., operation was performed on June 16, 1947. Resection of carcinoma of the sigmoid and aseptic end-to-end anastomosis was done. Exploration was carried out through the left mid-rectus incision which was about 4½ inches long. The bladder was tremendously dilated. A catheter was passed into the bladder and fully 2,000 cc. of urine were evacuated. The mass was in the mid-sigmoid about 4 inches above the lower peritoneal reflection. It was

about 2 inches long, seemed to encircle the bowel and there were a few dense masses in the mesentery immediately adjacent to the bowel. The remainder of the mesentery was free and the region of the growth itself in the sigmoid was not attached anywhere.

Exploration of the liver revealed two shotty, pea-sized nodules on the lower surface of the liver which seemed to be elevated. To palpation they seemed like typical metastatic nodules, but they were not visualized because of the exposure on the left side. There were no other masses in the liver and no other masses along the posterior lymphatics. Accordingly, resection of the tumor was done with immediate aseptic end-to-end anastomosis. The tumor was mobilized and the peritoneum on the outer and medial surfaces was incised. The ureter was easily seen. The spermatic vessels were seen and the growth was removed down to the superior hemorrhoidal vessels. This was a very large trunk and it was dissected cleanly. The bowel was divided about 1 inch below the growth and about 3 inches above the growth. The division was made over a Wolfson clamp and the bowel cut through and then cauterized. Immediate end-to-end suture was made over two crushing clamps, using other surface interrupted silk and inner chromic sutures. At the conclusion there seemed to be a very good anastomosis and the junction admitted the end of the thumb easily. The peritoneum was then sutured together both on the mesial side and on the lateral side. Apparently hemostasis was satisfactory. The wound was closed.

Pathological diagnosis: adenocarcinoma of sigmoid; metastases to adjacent lymph nodes and to epiploic fat.

Convalescence was fairly satisfactory. There was some ileus for the first few days but on the sixth postoperative day the bowels moved normally and continued to do so. The patient had to be catheterized regularly after the operation. The urologist (Dr. W. K. Rexford) diagnosed a mild cord bladder, symptomati-

cally present before the operation (x-ray appearance of enlarged bladder and dilated bladder at the time of the operation) and on June 27, 1947, a transurethral punch operation was performed. "There was some obstruction of the bladder neck. Three or four pieces of tissue were removed from the floor of the bladder neck. Bilateral vas ligation was done." There was very little reaction from this operation but suddenly on June 29, 1947, the patient began to have some diarrhea.

On June 30, 1947, he was uncomfortable with recurrent cramps. There was some distention toward evening, chiefly in the right lower quadrant where a vague sensation of a mass could be felt in the right rectus region. There was no spasm or rigidity of the abdominal wall. White blood cells were 12,000; hemoglobin, 14 Gm. = 91 per cent; red blood cells, 5.07. The temperature had gone up to 100.4°F. after the transurethral punch operation but had been 99.2 to 98.6°F. since then. There was also some evidence of shock, in that there was cyanosis, sweating and coldness of the extremities. Blood pressure was maintained satisfactorily. Preoperative diagnosis of recurrent intestinal obstruction was made and the patient was taken to the operating room on the evening of June 30, 1947.

A long McBurney incision was made just above the right crest of the ilium. On entering the abdomen some dark colored serous fluid was aspirated. There was a loop of very dark colored small bowel apparent toward the midportion of the abdomen from the incision. The incision was prolonged downward and upward, partially cutting the muscular fibers of the abdominal wall. It was then seen that there was a loop of bowel (small bowel) which was totally gangrenous. It was about 16 to 18 inches in length and ended sharply. At a sharp demarkation point of the bowel both proximally and distally the bowel was viable. The mesentery itself did not look too unusual. There was no marked dilatation either above or below this area and the large bowel was visible in the depth of the wound; this was not dilated. Evidently the condition was a localized mesenteric thrombosis. As far as exploration would allow no larger extent than has just been described was involved. The involved bowel was tense, leathery, engorged and practically black.

Ochsner clamps were applied through the viable bowel, both above and below the gangrenous portion. The mesentery of the gangrenous portion was excised between clamps. An aseptic anastomosis was then done over the two Ochsner clamps, interrupted silk for the outer layers and running No. 00 chromic for the inner layer. When the anastomosis was made, a good lumen was palpated through the anastomotic line and the anastomosis itself looked clean. A few sutures tacked the mesentery together and the abdomen was closed without drainage. The condition of the patient at the end of the operation was much better than it was before. The shock had disappeared, the extremities were warm and the pulse was quiet. He was placed on dicumarol therapy for one week.

Following this, there was still some trouble with abdominal distention; although his bowels moved five days postoperatively and he continued to pass gas and liquid stools, his abdomen was not normal. The Levine tube was replaced by a Cantor tube on the fifth postoperative day. Its progress down the intestinal loops was slow. Gas and feces would follow an enema and flatus would pass frequently, but large amounts of gas and small intestinal contents were always brought up with the Cantor tube. He had persistent extrarenal azotemia, i.e., he was drowsy and the blood nitrogen hovered around 70 to 50 and would not get lower. Urinary output was well maintained (1,000 to 1,500); chlorides, blood proteins, hemoglobin, red blood and white blood cells were normal.

After two weeks went by it was evident that although we could maintain him in electrolyte balance and his bowels moved, there was some partial obstruction of the small bowel which would not allow normal passage. If the long intestinal tube was clamped for two to three hours, he was uncomfortable and distended. If clamped more than three hours, he was tense and in pain. As soon as the clamp was opened he became rapidly deflated in two to three minutes with passage of considerable gas and liquid through the tube.

The patient was re-explored July 18, 1947. Exploration was done through a right midrectus incision. The bowel which presented was not dilated but the wall was thickened. The large Cantor tube was easily palpable in the lumen; it had completely deflated the bowel. The bag containing mercury was found about

3 inches above a tightly adherent area between the small bowel and the posterior peritoneum. The bag was about 3 inches long and about 1½ inches in diameter and had apparently absorbed some air from the bowel. The constricted and obstructed area was in the lower ileum. Here the small bowel was densely adherent to a tag of posterior peritoneum in the region of the promontory of the sacrum. This was dissected free, the bowel serosa repaired and the raw area which was left in the posterior peritoneal region tacked together. About 6 inches below this was the site of the end-to-end anastomosis. This was also caught tightly in a constricting band between the small bowel and the mesentery. When this was liberated, the end-to-end anastomosis was seen to be satisfactory. The finger easily invaginated in it and there was no obstruction. The raw areas here were repaired as well as possible. This area was about 8 or 10 inches from the ileocecal valve. A small piece of oxycel gauze was put against the raw area in the posterior peritoneum where the bowel had been adherent.

There was no exudate in the abdominal cavity and no discoloration or lack of luster of the serosa of the small bowel. The remainder of the abdomen was not explored. There was definitely no further advancement of the mesenteric thrombosis process so no exploration was done for any sign of further extension of the malignancy. The wound was closed in the usual fashion. The condition of the patient at the end was good.

From then on the patient's convalescence was satisfactory. He was discharged from the hospital July 30, 1947. His bowels were moving normally. He subsequently had a transurethral prostatectomy (December, 1947). When last seen on April 1, 1948, he was gaining weight, going to business daily, his hemoglobin was 88 per cent and his bowels moved without discomfort. He urinated easily every two to three hours, felt alert and well and had no complaints.

Whittaker and Pemberton[1] analyzed sixty cases of mesenteric thrombosis at the Mayo Clinic in 1938, and Laufman and Scheinberg[2] carefully studied forty-two cases in Michael Reese Hospital, Chicago in 1942. Their mortalities were 90 to 95 per cent. Thirty to forty per cent of the cases in each series followed operations; the list comprised chiefly splenectomy, stomach, biliary, pancreas, colon and gangrenous appendix operations. In Pemberton's series in only three cases, which followed operation was reoperation attempted. Only one survived; a patient who had a mesenteric thrombosis following hysterectomy. Sixteen of his other patients were operated upon for primary mesenteric thrombosis with only two survivals. Nine of Laufman's forty-two patients were operated upon with only three survivals.

As Laufman and Scheinberg pointed out there are several reasons for the high mortality in mesenteric thrombosis. The patient is very often moribund at the time it is recognized; auricular fibrillation, circulatory failure, etc. are common. The thrombotic process may involve the entire superior mesenteric vessels immediately, death ensuing before gangrene develops. The process of thrombosis frequently continues to extend and propagate itself after operation. Murray[3] believes that this is the principal reason for high mortality. Finally, there is termination in gangrene and peritonitis.

By and large the recovered patients have been those who have undergone resections of relatively small segments of intestine. Brown[3] in a review of the literature, estimated that removal of up to 3 feet of small bowel carried a hopeful prognosis; any more extensive involvement "sharply increases the mortality."* In our case the resected specimen measured 14 inches, well within this limitation. Laufman and Scheinberg suggested that larger quantities of blood than the usual 500 cc. are required in these cases and that perhaps more frequent use of larger quantities of blood (up to 2,500 cc.) would lower the mortality. Murray gave heparin postoperatively to six patients with mesenteric thromboses in

* Madding and McIntire[4] recently reported a case in which the entire small bowel except the upper 4½ feet of the jejunum and the entire ascending colon to the hepatic flexure was involved. This entire length of bowel was removed and jejunotransverse colostomy done. The patient recovered.

whom 1½ to 7 feet of bowel was removed. He believed that thereby propagation of the thrombus would be stopped. Four of these patients were living and well one year later, and it was the judgement of the operating surgeons that "none of these would have recovered." Much has been written recently about the usefulness of paravertebral novocain block in relieving the arterial spasm which accompanies venous thrombosis of the extremities. Edgar Allen[7] is critical of its value in these cases either for relieving spasm, except occasionally, or for stopping propagation of the venous thrombosis. Tetraethyl ammonium chloride has a similar paralyzing effect on the sympathetic and parasympathetic ganglia. A generalized body effect lasting six to eight hours can be obtained with an intramuscular injection. We have seen no series of cases reported since 1942 in which the previously mentioned advances in surgical care have been evaluated.

Regarding etiology, it is known that mesenteric thrombosis is prone to follow splenectomy when factors conducive to thrombosis are present, and it also follows abdominal suppuration or operations in which soiling has taken place. In our patient it followed colon resection and aseptic end-to-end anastomosis. Recent reports, although favoring aseptic end-to-end anastomosis when feasible, do not show statistically that there is any increased safety or lessened morbidity over so-called open anastomosis. In this latter technic there is bound to be some spillage, but the increased accuracy of suturing and the viability of a non-crushed bowel edge are compensating advantages. Whether the ileus our patient had for five days postoperatively, which was a bit more than usual, indicated slight peritoneal soiling is doubtful. He had no signs of cardiac trouble either before or after operation.

Regarding diagnosis, there is no uniformity to the pain in mesenteric thrombosis. "The pain is a true visceral pain, not associated with muscular spasm or tenderness of the abdominal wall. It differs from simple vascular pain only in its constancy."[6] Until or unless gangrene or peritonitis develops, there is little abdominal wall spasm.

Most observers believe that they cannot accurately tell venous from arterial mesenteric thrombosis except in general terms; venous occlusion tends to develop more slowly, pain and collapse are less. If a small segment is involved or venous thrombosis develops slowly, the pain is less. If the main artery is suddenly occluded, pain is agonizing, severe and there is considerable shock and collapse. Pain usually overshadows vomiting, as a differential point from intestinal obstruction. The pain of our patient was more of a crampy discomfort, constant but not severe. There was a vague mass. Distention was present but abdominal wall spasm was just beginning.

Regarding the operative indications in our patient, the second operation, at which mesenteric thrombosis was found, was done without delay. There was a tender mass and signs of beginning peripheral collapse. By contrast the third obstruction was operated upon only after eighteen days of careful observation and meticulous preparation. This delay paid dividends. It took more than one week for the long intestinal tube to find its way down the length of the small bowel, but since the patient's fluid, electrolyte balance, etc. were maintained there was no hurry. At the third abdominal operation the small bowel was completely deflated and although thickened it could be handled, sutured and adhesions dissected as if it were normal and not obstructed. Recent reports are bearing this out. The lowest mortalities are obtained when operation is not undertaken until the bowel has been completely deflated provided, of course, that strangulation can be ruled out.

SUMMARY

A case is described in which three successive operations for intestinal obstruc-

tion were done; resection and end-to-end sigmoid anastomosis for cancer, resection of the ileum for mesenteric thrombosis, and dissection and liberation of constricting adhesions for simple postoperative obstruction. The indications and clinical course especially as they pertain to mesenteric thrombosis are briefly discussed.

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MACALPINE reviewed nineteen cases of papilloma of the renal pelvis and found that diagnosis was facilitated by finding tumor cells in the urine and a growth projecting into the ureter with a characteristic filling defect. Clinically, the condition often simulates hypernephroma. Removal of the kidney and of the whole of the ureter flush with the bladder is the recommended treatment. If the renal pelvis is opened, papillomatous tissue may escape and become transplanted and grow, causing future complications. In four of the reviewed cases this occurred and such transplants assume malignant characteristics. Early diagnosis and complete ureteronephrectomy are therefore necessary if one is to obtain satisfactory results. Hematuria is often an initial symptom in these cases of papillomatous disease. (*Richard A. Leonardo, M.D.*)

New Instrument

SUBPERIOSTEAL PERICOSTAL BAND FOR THORACIC CAGE APPROXIMATION

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THE problem of closing the chest following intrathoracic surgery is often associated with many difficulties. In

ing the intercostal nerves. For this reason many thoracic surgeons have abandoned this method and are suturing only the
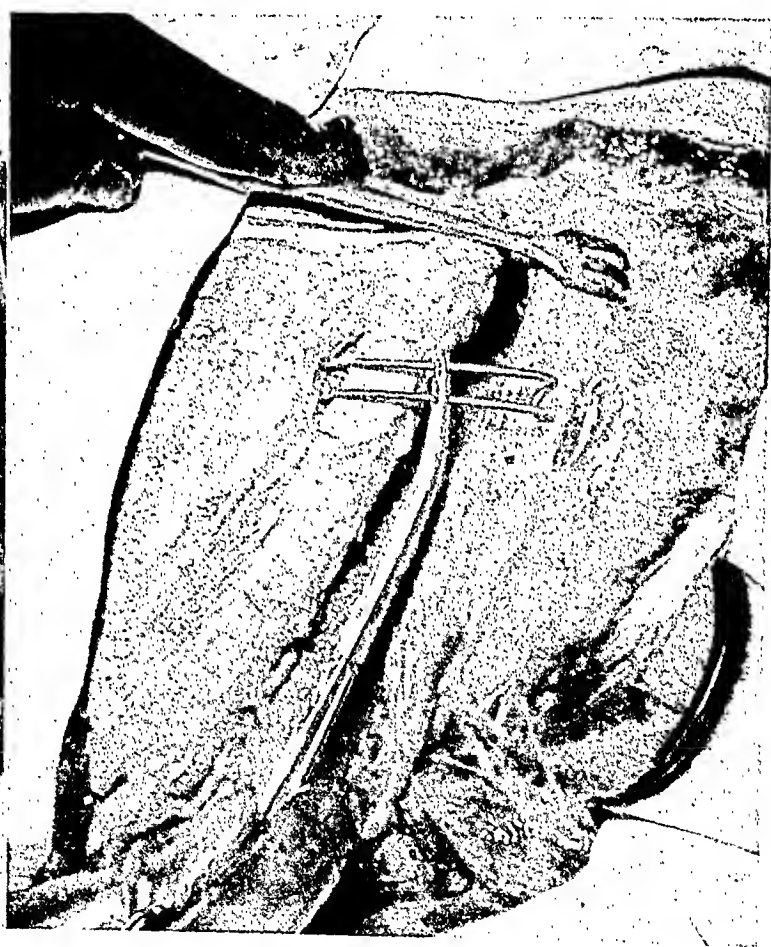
FIG. 1. Photograph illustrating removal of segment of periosteum.
FIG. 2. Technic for applying band.

former years the method of closure went through the same periods of trial and error as confronted the abdominal surgeon. Thus, the thoracic surgeon has passed through the era of catgut, silk and, finally, stainless steel wire. The methods have been far from ideal. These ligatures have been applied extraperiosteally without consider-

periosteal bed. Some men employ pericostal sutures; however, they place them beneath the periosteum. This method[1] was suggested by the author and has the advantage of eliminating postoperative pain in the chest wall due to compression of the sensitive periosteum and intercostal nerves by the encircling sutures.
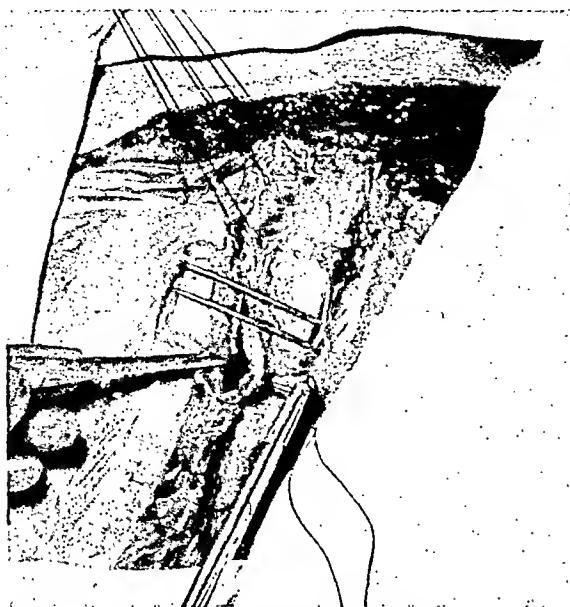
FIG. 3. Band *in situ* with interrupted silk closure of periosteal bed.

As a new method of closing the thoracic cage which is an improvement over former methods, including the procedure already mentioned, the author has employed a metal band. Its use not only successfully prevents postoperative pain but, in addi-

tion, has many other advantages. These advantages are: (1) It is very simple to apply. (2) It diminishes the time required for emergency closing of the chest cavity. (3) The use of this approximator necessitates the removal of only small segments of the periosteum. (4) It is a rib approximator which obviates the necessity of employing special instruments and additional assistants to hold the ribs in approximation for suturing. (5) Its steel construction results in a strong chest wall which cannot separate and thus prevents the possibility of wound disruption. (6) The resulting chest wall enables the patient, immediately following the operation, not only to breathe normally but also to turn from side to side and cough if necessary without fear of wound dehiscence and thereby the natural expulsion reflex is preserved. (7) When employed properly, the band does not impinge upon the intercostal nerve and prevents irritation of the sensitive periosteum.

This metal band is constructed of stainless steel and made in varying lengths which have been numbered from one to

FIG. 4. Postoperative x-ray illustrating the clip.

five.* Number one corresponds to 3 cm. and number five corresponds to 5 cm. All are of uniform width. These clips are applied manually while the assistant holds the ribs to be approximated with rake retractors. When this has been accomplished, the surgeon then approximates the soft tissues of the chest wall. (Figs. 1 to 3.)

This method of closing the chest wall has been employed on many occasions with great ease. Following this procedure, patients have no discomfort. It has never been employed in children because the thoracic cage has not reached its maximum growth and the presence of the clip may influence proper anatomic and physiologic growth. In the event that it becomes necessary to remove the clip, this may be done under local novocaine anesthesia with minimal discomfort or distress to the patient. However, the occasion has never presented

* Manufactured by The George P. Pilling and Son Co., Philadelphia, Pa.

itself which necessitated the removal of the metal band.

CONCLUSIONS

A new method is presented for closing the operative wound following intrathoracic surgery. This method employs a metal band of stainless steel of varying lengths. Usually one or two clips are sufficient for approximating and maintaining the ribs. The advantages of this method have been enumerated. It is realized that some disadvantages will arise in the future. However, up to the present time the metal clip has proven to be most satisfactory.

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AUTHOR INDEX TO VOLUME LXXV

SUBJECT INDEX TO VOLUME LXXV

(E.) = Editorial